Indoor environment and the impact on the health of pre-existing asthmatics at work – the development of a risk management framework.

Valerie Cameron

This thesis is submitted in partial fulfilment of the requirements of the Abertay University for the degree of Doctor of Philosophy

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I certify that this is a true and accurate version of the thesis approved by the examiners

Signed_____ **Date**_____

(Director of Studies)

Declaration

I, Valerie Cameron, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I can confirm that this has been indicated in the thesis.

Signed_____ **Date**_____

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Abstract

This thesis examines the spatial and temporal variation in nitrogen dioxide (NO_2) levels in Guernsey and the impacts on pre-existing asthmatics. Whilst air quality in Guernsey is generally good, the levels of NO_2 exceed UK standards in several locations. The evidence indicates that people suffering from asthma have exacerbation of their symptoms if exposed to elevated levels of air pollutants including NO_2, although this research has never been carried out in Guernsey before. In addition, exposure assessment of individuals is rarely carried out and research in this area is limited due to the complexity of undertaking such a study, which will include a combination of exposures in the home, the workplace and ambient exposures, which vary depending on the individual daily experience.

For the first time in Guernsey, this research has examined NO_2 levels in correlation with asthma patient admissions to hospital, assessment of NO_2 exposures in typical homes and typical workplaces in Guernsey. The data showed a temporal correlation between NO_2 levels and the number of hospital admissions and the trend from 2008-2012 was upwards. Statistical analysis of the data did not show a significant linear correlation due to the small size of the data sets.

Exposure assessment of individuals showed a spatial variation in exposures in Guernsey and assessment in indoor environments showed that real-time analysis of NO_2 levels needs to be undertaken if indoor micro environments for NO_2 are the be assessed adequately. There was temporal and spatial variation in NO_2 concentrations measured using diffusion tubes, which provide a monthly mean value, and analysers measuring NO_2 concentrations in real time. The research shows that building layout and design are important factors for good air flow and ventilation and the dispersion of NO_2 indoors.

Environmental Health Officers have statutory responsibilities for ambient air quality, hygiene of buildings and workplace environments and this role needs to be co-ordinated with healthcare professionals to improve health outcomes for asthmatics.

The outcome of the thesis was the development of a risk management framework for pre-existing asthmatics at work for use by regulators of workplaces and an information leaflet to assist in improving health outcomes for asthmatics in Guernsey.

Acknowledgements

I would like to acknowledge all colleagues, friends and family who have assisted me with this research. Without their support, I would not have completed this work.

In particular, I would like to thank, most wholeheartedly, my supervisor at Abertay University (AU), Dr Kehinde Oduyemi, who supported me through this six year part-time programme and who was always at the end of the telephone or on email to provide me with inspiration. It was by chance that we were discussing the impacts of indoor air quality, when the idea for this research was formulated. That serendipitous conversation made a huge impact on my life over the last six years and has changed how I think about things.

I would also like to thank Dr David Blackwood, Dr Philip Colliers and the research team at AU for supporting me and keeping me on track. Their support has been invaluable for an off-campus student.

I wish to thank all of the people who helped me to collect the data for my research, especially the staff at the Princess Elizabeth Hospital in Guernsey, for providing access to data and for allowing their workplaces to be monitored as part of the diffusion tube survey. I thank Jenny Elliot (Senior Clinical Coder) and Jenny Cataroche (Epidemiologist) for their assistance.

I am grateful to the Guernsey Public Health team, who organised the clinical lunch training sessions, for allowing me to present various aspects of my research on several occasions as part of the Continuing Professional Development programme for the Health and Social Services Department.

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I also thank the people who opened their homes for part of the diffusion tube survey and for allowing me to make plans of their properties. Especially, I thank Mr and Mrs T Rowe and Mr and Mrs P Goodchild.

The survey of Scottish Local Authority staff took place after permission was granted by the Chief Officers of those departments. I thank the Society of Chief Officers of Environmental Health in Scotland for giving their support to this research, and also to the staff of the 13 Scottish Local Authorities who responded to the survey. I am very grateful to you all and hope the outcomes of the survey assist you in developing better interventions for asthmatics at work.

I would like to thank the staff at Environmental Scientifics Group Laboratories in Didcot, in particular Andy Parish and Trevor Worrall for allowing me to visit their laboratories to gain a better understanding of diffusion tube preparation and techniques for their analysis.

Most of all, I thank my husband, Stephen Cameron, for the long years of encouragement and for constantly driving me forward to complete this project. Also for the cups of tea and the smiles that kept me going throughout.

I have been passionate about asthma for many years as both of my children were diagnosed as asthmatic as infants. On many occasions, over the years, they struggled for breath during asthma attacks and were admitted to hospital where they were treated by many amazing doctors and nurses in the NHS, who assured their survival, and to whom I am deeply grateful.

I therefore, dedicate this work to my son and daughter, Adam and Ruth Kinniburgh who, still in adulthood, suffer the consequences of this debilitating condition and who inspired me to undertake this research project.

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List of abbreviations

AU	**Abertay University**
BTX	**Benzene, Toluene and Xylene**
CIEH	**Chartered Institute of Environmental Health**
CO	**Carbon monoxide**
DRP	**Disease reduction programme**
EC	**European Community**
EHO	**Environmental Health Officer**
ERS	**European Respiratory Society**
ESG	**Environmental Scientifics Group**
EU	**European Union**
Fit3	**Fit for work, fit for life, fit for tomorrow – HSE strategy delivery programme**
GP	**General Practitioner**
HASCOG	**Health and Safety Scottish Coordinating Group**
HPS	**Health Protection Scotland**
HSE	**Health and Safety Executive**
ICD 10	**International Statistical Classification of Diseases and Related Health Problems – volume 10**
NAQS	**National Air Quality Strategy**
NHSHS	**NHS Health Scotland**
NO_2	**Nitrogen dioxide**
NO_X	**Oxides of nitrogen**

OEHPR	**Office of Environmental Health and Pollution Regulation**
OPA	**Operational partnership agreements**
PEH	**Princess Elizabeth Hospital**
PM_{10}	**Particulate matter 10 microns or below in size**
SO_2	**Sulphur dioxide**
REHIS	**Royal Environmental Health Institute of Scotland**
SOCOEHS	**Society of Chief Officers of Environmental Health in Scotland**
UKAS	**United Kingdom Accreditation Service**
WHO	**World Health Organisation**

Chapter 1

Introduction to the Thesis

Overview

This chapter describes the purpose and focus of the research. It contains a review of the current literature and knowledge about asthma, the environmental conditions that can exacerbate respiratory symptoms and the risk management of those symptoms in workplaces.

There is a huge body of evidence relating to ambient (outdoor) air pollution and its impact on respiratory health and this is generally used as a key indicator of respiratory health in national health policy. This chapter explores this in relation to multiple exposure assessment and will examine whether ambient air quality should be used as a proxy for the respiratory health for asthmatics in national policy which will direct the delivery of actions and interventions by regulators.

The review of the literature will bring clarity to the original hypothesis and will shape the research aims and objectives, which are summarised at the end of the chapter.

This topic is diverse and complex and, in order to maintain focus, a ‘literature review strategy’ was drawn up and this is included in Appendix 1. It should be noted that the Appendix numbers relate to the Chapter numbers, so supporting material for Chapter 1 is found in Appendix 1 and so on.

Following the literature review, a detailed plan of the thesis was developed and this is shown in section 1.5, figure 1.5(i).

1.1 Purpose of the Thesis

The purpose of this research is to explore the factors that exacerbate asthma in pre-existing asthmatics in indoor environments. Of particular interest is the risk management of pre-existing asthmatics in the workplace and how their working life can be improved by the development of a risk management framework for use by employees who suffer from asthma, their employers and workplace regulators.

Asthma is a debilitating ill-health condition caused by the inhalation of an allergen which causes an allergic reaction. The symptoms that follow include sneezing, coughing, wheezing and shortness of breath, which can be mild or more severe and can occasionally lead to death. There are three variations in symptoms: these are airway obstruction, inflammation and airway irritability (WebMD, 2008). Airway obstruction happens when the muscles in the airways tighten following environmental or allergenic triggers as described later in this section. Inflammation of the airways happens when the bronchioles swell and restrict inhalation and airway irritation is the consequence of the sensitisation of the airways following allergen inhalation and may lead to lung damage over time.

A huge amount of research has been undertaken into the impacts on respiratory health from ambient (outdoor) air pollution such as particulates and gases from road traffic emissions and industry (Beverland, 2007; Prescott, 2000). The evidence indicates a direct inverse correlation with the spatial and temporal levels of pollutants and the respiratory health of the local population. As the level of air pollutants increase, the respiratory health of the population decreases (Di Giampaolo et al, 2011).

It therefore follows that interventions in the environment to improve air quality will bring about a direct improvement in the health and wellbeing of asthmatics, and the respiratory health of the wider population.

The effect of indoor environments and air quality is now being recognised as a significant impact on health. It is estimated that people spend as much as 80% of their

lives indoors (WHO, 2002), at home, at school, at leisure or at work. Studies have shown that ambient air pollutants can be found indoors and so it can be assumed that if ambient air pollution levels rise, then this will also increase indoors, although indoor air quality is rarely monitored, assessed and characterised so the relationship between ambient air quality and indoor air quality requires further assessment.

Whilst there is a direct correlation with hospital admissions for respiratory disorders with increases in ambient air pollution, this may well be contributed to by other exposures indoors. The research will examine the correlation between hospital admissions for asthma and air quality measurements recorded in Guernsey from 2008-2012 to establish whether, or not, there is a correlation, both spatially and temporally. This work has never been done in Guernsey and will assist in the development of an air quality strategy and legislation for that jurisdiction. It should be noted that the UK National Air Quality Strategy (NAQS) is considered as a benchmark in Guernsey but is not legally binding.

This research will identify the allergic mechanisms involved with asthma, and will assess the current thinking on prevention and control to bring about improvement in the health and well-being of asthmatics through effective risk management of key risk factors.

Asthmatics experience episodes of ill health after being exposed to an ‘asthmagen’ that triggers an immune system response, or ‘asthma attack’ (Asthma UK, 2013; House of Lords, 2007). This may be as simple as a bout of wheezing or may be result in constriction of the airways to the point of respiratory collapse. In order to assess the total exposure of an individual it is essential to consider all exposures. Therefore, ‘human exposure assessment’ is a crucial facet of risk management of asthmatics when considering workplace exposures, which cannot be assessed in isolation.

Many studies to date concentrate on medical interventions (Royal College of Physicians, 2003; Bsaci, 2013; Camargo, 2009; Department of Health, 2012) however, the contribution made by local authority Environmental Health Professionals, in prevention and control of environmental stressors, is often unnoticed and unrecorded.

This research will, for the first time, include the role and function of local authority Environmental Health Officers (EHOs) in risk managing asthma at work.

For many years, local authority Environmental Health teams have been involved in meeting the objectives of the NAQS in reducing ambient air pollution that causes and exacerbates respiratory disorders although the NAQS has not, to date, considered the assessment of indoor air quality.

This research will examine the role and function of Environmental Health Professionals in the prevention and control of asthma through interventions on indoor workplace environments. However, other environments will also be assessed as they may contribute to the total exposure experienced by working individuals. It is assumed that the workplace will contribute the major exposure in a 24 hour period, followed by exposures in the home. Workplace risk assessment is usually carried out following statutory guidance from the Health and Safety Executive (HSE) which provides guidance on assessment of health risk and safety risk of those at work. However, this guidance is generic and is not disease/condition specific so the process of determining the level and scale of health risk and safety risk may be open to individual interpretation. A risk management framework will, therefore, be developed from the research to ensure that this serious ill-health condition is managed effectively and consistently.

1.2 The Research in Perspective

There are around 6 million asthma sufferers in the UK and according to Asthma UK (2008) 90% of people suffering with asthma attribute their asthma to inhaled dust (which includes pollen etc.). Many asthma sufferers report worsening conditions when exposed to air pollution from industry and vehicles, particularly particulate matter and gases (Lin et al, 2004). The UK ranks the highest in the world for symptoms associated with asthma and it is thought that, although there may some genetic factors to consider,

most of this is caused by increased exposures to allergens and exacerbation by inhalation of air pollutants (The Global Asthma Report, 2011).

The NHS (2012) reported that 1000 deaths per year were associated with asthma and of those 90% were preventable. Asthma UK stated that 'asthma hospitalises someone every seven minutes' (Asthma UK, 2013). This is a significant and largely preventable problem if the triggers and symptoms are effectively risk managed.

It is estimated that around 13 million working days are lost per year and that asthma has an impact on the economy in the order of about £2.3 billion per year (HSE, 2006).

During the 1980s and 1990s as many as 1 in 5 children in the UK were diagnosed as being asthmatic. More recently the Royal College of Physicians (2003) reported that 32% of 13-14 years olds in the UK were being treated for symptoms. The incidence of asthma has increased three-fold over the last twenty years. Infant studies show that symptoms of asthma increase when traffic emissions increase (Andersen et al, 2008). This will be primarily due to added exposures to particulates and nitrogen dioxide (Nishimura, 2013). Whilst some children 'grow out of' the condition as their bodies grow, 1 in 8 teenagers leave school with asthma and require long-term medication to control their symptoms. These young people, with *pre-existing asthma*, are now entering the workforce and it is uncertain how their condition is risk assessed and risk managed while they are at work.

It is thought that 9-10% of adults seek treatment for the symptoms of asthma and around 35% of those with asthma had an asthma attack in the previous year (NHS, 2012). Although similar data does not exist currently in Guernsey, population health trends tend to mirror UK trends for most ill-health conditions and diseases.

As most adult individuals spend around 8 hours a day at work, exposure in the workplace will be a significant part of their daily exposure and therefore this warrants further investigation to develop interventions to minimise those exposures.

The HSE has researched extensively into 'occupational asthma' i.e. asthma caused *for the first time* by exposures in the workplace, and the essential risk assessment and risk

management of such exposures. This identified that those involved in wood working, flour mills and bakeries, and the use of isocyanates, aldehydes, latex, adhesives etc. resulted in the most cases of occupational asthma. Their research does not determine, however, the same controls for the risk assessment and risk management of pre-existing asthmatics in the workplace. Research by the HSE: *The true cost of occupational asthma in Great Britain*: was based on the prevalence of asthma in the work force. However, this calculation did not distinguish between pre-existing asthmatics and the incidence of new cases caused by workplace exposures. For the first time, the cost of occupational asthma was determined. The converse, however, was that the cost of work days lost, cost of treatment etc. for pre-existing asthmatics was not calculated separately at that time.

1.2.1 What is asthma?

Asthma is a chronic inflammatory disease of the airways and is caused by an immune response to an allergen, causing the release of an allergy antibody - Immunoglobulin E (IgE). The release of IgE is now thought to be the most important predisposing factor in the development of asthma (US Expert Panel Report, 3:2007). If released, IgE binds to receptors on mast cells, found in all tissues that have contact with the external environment, in this case the connective tissues of the lungs and the airways. This combination of IgE and mast cells allows the body to identify the allergen again if there is another exposure to the allergen. This is known as ‘sensitisation’. When the combined mast cell is then exposed to the allergen during another episode, they become ‘cross linked’ and the mast cell is activated to release inflammatory chemicals, such as histamines. The inflammatory response is often associated with increased releases from mast cells and mucous production in the airways and therefore further restriction of the already inflamed and narrowing airway, leading to wheeze and coughing (Carroll et al, 2002). This is known as an ‘IgE mediated allergic response’. Figure 1.2.1(i) illustrates this process.

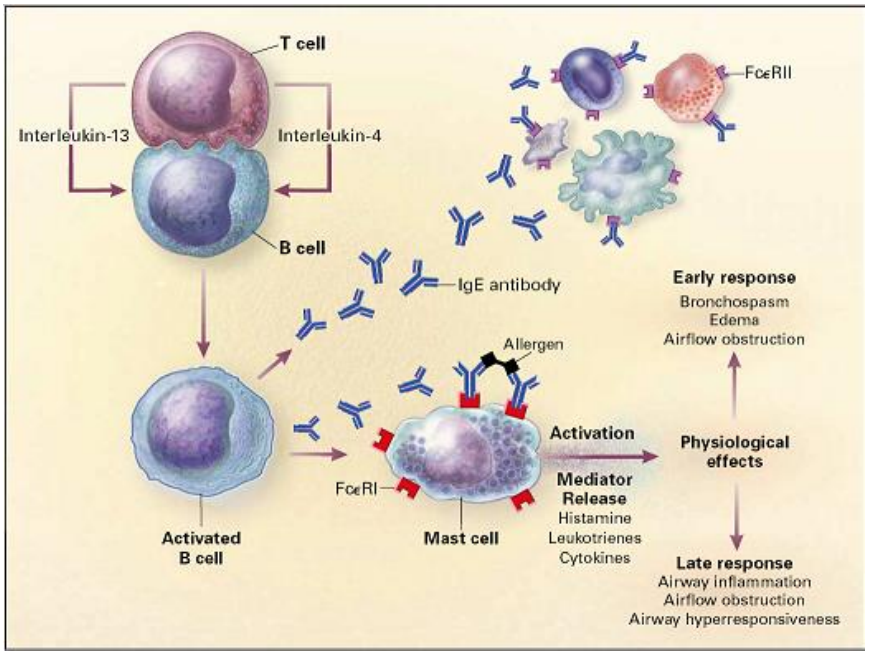


fig.1.2.1(i) allergic response causing release of IgE – (WebMD 2009)

The symptoms that follow include sneezing, coughing, wheezing and shortness of breath, which can be mild or more severe and can occasionally lead to death. Figures 1.2.1(ii) and 1.2.1(iii) show the constriction of the airways during an asthma attack and 1.2.1(iii) shows areas of mucous production (Cameron, 2009).

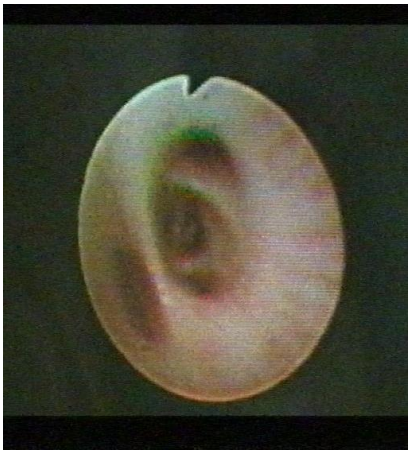


fig.1.2.1(ii)

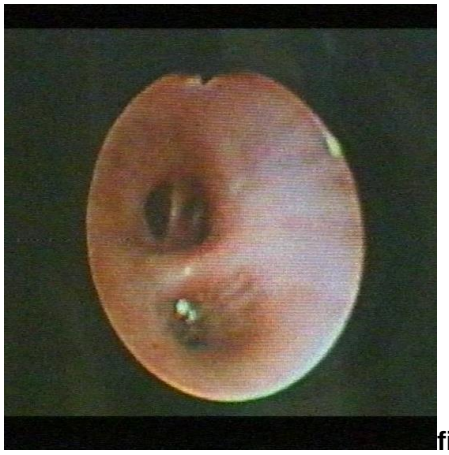


fig. 1.2.1(iii)

Figures 1.2.1(ii) and 1.2.1(iii) showing constriction of the airways and mucous production during and asthma attack (slides extracted from footage of BBC documentary 'Asthma' 2007)

Constriction of the airways and mucous build-up leads to the wheeze, coughing and breathlessness of the sufferer which typifies asthma (Abramson et al, 2013).

Work undertaken by the HSE in 2008 examined the difference between 'irritancy' and 'sensitisation' in workers at five different locations. Workers who had been exposed to allergens (latex and insect allergens) in their workplace and those exposed to irritants (welders and metal workers) all displayed the symptoms of coughing and wheezing that typifies asthma. However the study showed that exposure to respiratory irritants, in this case gases released during welding and metal processing, caused coughing etc. but did not bring about sensitisation i.e. repeated recognition of an allergen leading to the release of histamines or cytokines and the IgE immune system response. The presence of an irritant gas in combination with allergen exposure, however, lead to a more significant response i.e. exacerbation of symptoms (Ayres et al, 2004).

Casper (2008) showed that cheese workers who were exposed to air-borne enzyme powders used in cheese making became sensitised and developed occupational asthma. Once sensitised the workers displayed symptoms of coughing and wheezing if exposed again.

According to Camargo (2009) "*Asthma exacerbations consist of acute or subacute episodes of progressively worsening shortness of breath, coughing, wheezing, and chest tightness or any combination thereof*". Therefore once a person has been sensitised to an allergen they react again when exposed in the future, the ensuing symptoms may be made worse by additional factors, such as air pollution, if experienced at the same time.

Whilst the prevalence of asthma is on the increase the number of deaths attributed to asthma is now declining due to improved treatment and education of sufferers. (Chinn, 2004; US Expert Panel Report, 3:2007). In the UK quality standards for the management of asthmatics have been introduced by the National Institute for Health and Care Excellence (NICE, 2013) which, if implemented by clinicians, ensure the rapid management of symptoms and dramatically reduces the risk of death in patients.

1.2.2 Allergy and Allergens

The term allergy was introduced in the early 1900s to describe how the immune system was generally affected by foreign proteins, although nowadays, the term allergy is used to describe conditions that lead to various levels of sensitivity and adverse reactions to foreign proteins in the body following exposure to allergens.

Allergy in the UK is reaching ‘epidemic proportions’ (House of Lords, 2007) and this includes all allergies that impact on health of the population. Not only do allergies impact on the health and wellbeing of individuals, there is also an impact on their daily life, their families, their workplace and the economy. Whilst there are a range of allergens that cause reaction and irritation to sufferers, such as food allergies, this study is concerned only with inhaled allergens and air pollution.

Often the inhalation of grass and flower pollens, tree spores, dampness moulds and indoor dust, including the effects of exposure to pet dander or the faeces of house dust mites, lead to allergic reactions in those who are sensitive. It can be seen that there are a wide variety of allergens and some people have allergic reactions to individual allergens and some to combinations of allergens (MedicineNet, 2011).

Allergies and asthma attacks are compounded by the interaction between various allergens and environmental exposures, sometimes in complicated ‘cocktails’, making risk management of individuals extremely difficult (ISAAC, 1992; Bsaci, 2013; House of Lords, 2007). The symptoms of asthma can have many ‘triggers’ and these can affect individuals in different ways. For some individuals atmospheric pollution can trigger asthma symptoms, as can animal hair, pollen, poor, damp weather conditions, stress, energetic exercise, certain food ingredients, viral infections etc. therefore understanding their own trigger(s) is an important risk management issue for asthmatics (The Parliamentary Office, 2000; NHS Choices, 2013). Once an asthma attack has been triggered, i.e. the symptoms of wheezing, coughing etc. have started, certain factors like air pollution can increase, or worsen, the symptoms and this is known as ‘exacerbation’ (Nicholson et al, 1993).

This study will concentrate on the exacerbation of asthma symptoms attributed to air pollution, which can be reduced and controlled through strategic interventions and regulation (House of Commons, 2009-2010).

Unlike seasonal grasses, spores and pollens etc. found outdoors, indoor allergens can have an effect on asthmatics all year round (Allergy UK, 2013). Symptoms are more likely to be triggered and exacerbated during the winter when the weather is cooler and damp or during episodes of foggy weather (Price, 2007) and during the winter buildings are less likely to be well ventilated allowing the development of indoor pollutant micro-environments.

The House of Lords report in 2007 highlighted that about 25% of all ill health from allergies was caused by preventable environmental sources, and the important role of planning and environmental controls was underestimated. The report did not go on to explain what those planning and environmental controls should be and how they should be implemented. There was, in that report, also no mention EHOs in relation to the National Air Quality Strategy (NAQS) and workplace interventions for inhaled allergens and control of ambient air quality. This is a significant area of intervention that was missed by the report, potentially due to the lack of research and evidence in the area of local authority intervention. The role of EHOs in improving environmental hygiene, air quality and workplace risk management is often missed and joint approaches and interventions between medical and clinical practitioners in the NHS and EHOs in local government rarely happens in practice at local level (Cameron, 2003). Research into organisational culture indicated significant barriers and challenges to bring about joint working between local authority EHOs and NHS colleagues as part of the Community Planning process (Cameron, 2004). Fortunately in Guernsey the Environmental Health service is directly aligned with the health service and such barriers should not exist. In order to tackle the problem of increasing prevalence of asthma there will need to be a coordinated approach to policy and strategy and the ensuing preventative interventions delivered at local level. The forthcoming States of Guernsey report on Air Pollution will see, for the first time, a new regulatory regime aimed at tackling respiratory disorders by

interventions on the environment and this thesis will provide underpinning evidence for that report.

1.2.3 Human Exposure Assessment

The assessment of exposure to respiratory allergens is crucial in preventing and controlling ill health in pre-existing asthmatics. A number of exposures may contribute to exacerbation of symptoms experienced by asthmatics, however, the association of such risk factors and asthma is difficult to determine due to the intermittence of symptomatic episodes (Anto, 2004). Whilst the measurement of exposures helps to determine what people are exposed to, the actual dose inhaled will determine the health effect and ensuing symptoms.

Inhalation exposure to pollutants occurs in a multiple of micro-environments and the majority of that exposure occurs indoors (Weisel, 2002). Exposure to pollutants varies with time and location and can be influenced by the persons breathing rate and level of physical activity (Lioy, 1990), it therefore follows that an asthma sufferer working in a sedentary job is less likely to suffer an asthma attack at work, than someone with a heavy job and elevated respiration rate, environmental conditions being equal.

When evaluating air pollution data it can be seen that there can be fluctuations in concentrations hour by hour and the 'hourly mean' is often recorded along with a monthly and annual mean. Whilst the monthly or annual mean gives an indication of the general state of air quality, it may not be the best indicator for asthmatics who may be more susceptible to short-term temporal peaks and variations. Therefore individual exposure assessment is vital for individuals suffering from asthma. Whilst this phenomenon has been identified, there is little evidence that any interventions have been developed to support improvement.

In 2002 the World Health Organisation (WHO, 2002) considered the role of *human exposure assessment* in Air Quality Management, which led to the European Concerted Action on 'Urban Air, Indoor Environment and Human Exposure'. The committee acknowledged that exposure assessment plays a major role in risk assessment and risk

management and is "*a more direct environmental health risk indicator than ambient air measurements*".

The 2002 WHO workshop considered two approaches to exposure assessment:

1 Ambient exposures: using emissions inventories, dispersion modelling, air quality monitoring to give a detailed description of concentrations in time and space.

2 Indoor assessment: based on personal exposures and 'micro-environment' concentrations, apportioned to sources based on activity/behaviour and time.

Therefore human exposure assessment must take account of all temporal and spatial exposures (Delfino et al, 2008). For example, in a 24 hour day, there will be a multiple of exposures from the home, outdoors, the workplace and other indoor spaces used during that time period. The exposure to a pollutant will need to be measured in each space for the time spent there to give an accurate exposure. This is rarely carried out due to the complexity of the task, which involves measurements taken in the home, the workplace, other indoor places and the outdoor environment. Studies have been carried out separately e.g. in the home (Brugge et al, 2003) but combining all exposures is daunting and rarely undertaken.

The United States Environmental Protection Agency (US EPA) has been developing an exposure model for individuals (EMI) which recognises that ambient air quality measurements '*do not necessarily reflect personal exposures since pollutant concentrations observed indoors (e.g. homes, offices, factories, schools motor vehicles) can differ from those observed at central-site air monitors, and considerable time is spent by people in these indoor locations.'* The US EPA has therefore developed a model which accounts for the time-locations-concentration for individual exposures.

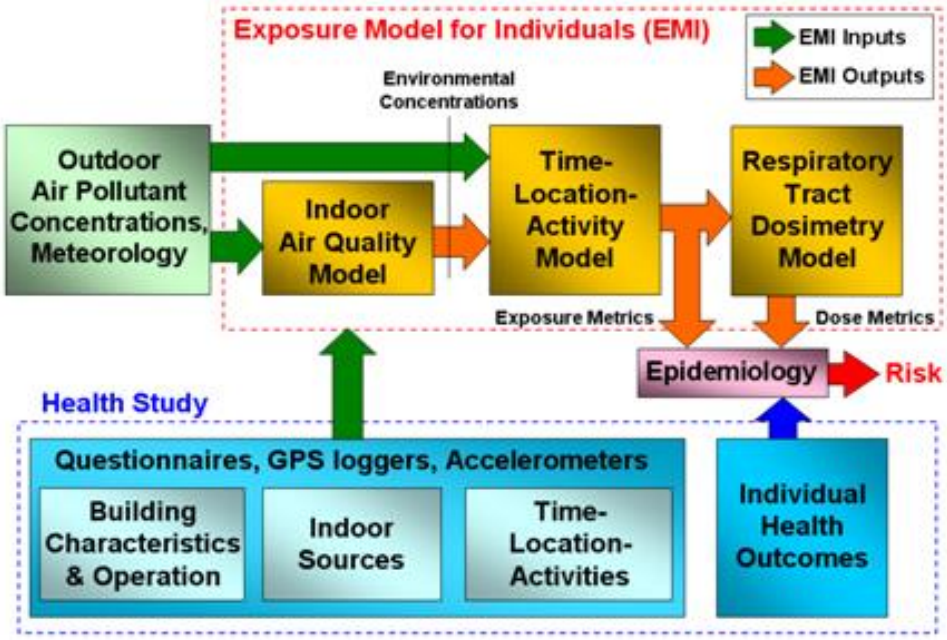


fig.1.2.3(i) Exposure model for individuals – US EPA (Breen et al, 2012)

Figure 1.2.3(i) identifies that outdoor air pollution and weather conditions are important inputs to the exposure model for indoor air quality and that the exposure of an individual relies on the amount of time spent in each location and the activity being carried out therein. This model also examines respiratory tract dosimetry as an input to epidemiology. Building characteristics, indoor pollution sources and again, time-location-activity are inputs to the indoor air quality. However, the model does not clearly indicate what the 'individual health outcomes' input to epidemiology means as indicated by the blue arrow, or how the inputs to epidemiology lead to risk as indicated by the red arrow. Epidemiology is generally the study of health and disease in the population, so it is unclear why it would be included in the 'exposure model for individuals'.

Exposure assessment relies on the measurement and evaluation of concentrations of pollutants and the risk assessment requires comparison with standards that are set to protect human health. In general terms though, these standards are set to protect the wider population and may not necessarily provide sufficient safeguards for those with pre-existing conditions like asthma. This poses a significant problem for those involved

in the risk assessment process e.g. local authority EHOs, who use such standards when carrying out various risk assessments.

"Inadequate human exposure classification continues to be one of the most pressing issues to be addressed in future environmental epidemiology research, and has important implications for both local government environmental health and central government environmental pollution monitoring policies." (Beverland, 2007).

1.2.4 Developing policy and standards for indoor air quality

In order for regulators to undertake their normal controls and interventions to protect health and wellbeing, there is usually a long process of research and policy development before any interventions actually take place. Regulation is the process of inspecting and auditing activities against a set of legislative standards and statutory guidance to ensure compliance.

Policy is developed at global, continental, national, regional and local levels. When policy is developed, there are often standards and strategic interventions, some statutory, that need to be agreed to ensure the policy is delivered and to bring about the necessary change for improvement. Standards are required so that there can be a measurement of success or achievement of interventions introduced by the policy e.g. an improvement in population health or improvement in environmental conditions.

Many policies are researched and developed at WHO level and are then cascaded into national jurisdictions or in the case of the UK, via the European Union in the form of EU Directives and EC Regulations. In 1997, the UK introduced its NAQS and set specific objectives for the achievement of standards for various ambient air pollutants, aimed at improving air quality through interventions on traffic emissions and introduced local air quality management areas where the standards were not being met. (The current NAQS objectives and standards are found in Appendix 1).

This is not reflected in the development of policy and standard setting for indoor air quality.

The WHO considered that policy developed by regulatory authorities needs to mitigate the indoor exposures. These should include changes in conditions e.g. traffic management, urban planning for ambient air quality management, but also measures to change individual behaviours e.g. the behaviour of residents indoors, including tobacco smoking. Regulation of an individual's behaviour in their own homes is an impossible task and change in that respect will only be brought about by better building design and education (WHO, 2002). From this assertion, building design is an important aspect that may be influenced by policy and statute to ensure improvements in indoor air quality. This will include design, layout, building materials, indoor airflows and ventilation.

It will be impossible for EHOs at local level, to implement controls over personal behaviours in the home, and so interventions on building design and layout are more likely to be successful strategies. EHOs are usually statutory consultees to the planning process and can therefore influence the building design process, although it is unknown whether consideration of indoor air quality and asthma is assessed during that process. Current statutory controls by EHOs tend to extend to improving ambient air quality and managing risks to health and safety in the work place. Control of ambient air quality and workplace exposures will address a large proportion of individual exposures to workers but does not address the risks to those who do not work. This will vary greatly between individuals, adding to the complexity of any policies developed at population level.

Council Directive 89/106 EEC (1989) introduced specific measures relating to construction products and particularly Annex 1.3 on hygiene, health and the environment where a building should be "*designed and built in such a way that it will not be a threat to the hygiene or health of the occupant or neighbours, in particular as a result of any of the following:-*

the giving off of toxic gas,

presence of dangerous particles or gas in the air,

emissions of dangerous radiation,

pollution or poisoning of water or soil, or

the presence of dampness."

These provisions are significant in respect of controls for asthmatics and yet are rarely considered in this context.

The WHO committee (WHO, 2002) went on to develop a model that identified the four tenets of public health (exposure, health effect, health impact and measures) in relation to risk assessment and risk management. Effective risk reduction strategies for the indoor environment depend on assessment of risks in micro-environments, activities and emission sources and so these will be important criteria of the development of the risk management framework. Micro-environments occur in buildings due to the ability of the fabric to 'filter out' or emit pollutants; layout and design where features such as location of windows, chimneys, staircases affect air circulation and ventilation; and impacts from combustion sources such as cooking and heating appliances. Indoor air flows play an important part in ensuring good ventilation and reduction in increased concentrations of pollutants.

The WHO Working Group (WHO, 2006) on the development of WHO Guidelines for Indoor Air Quality agreed that indoor air quality was a significant determinant of health, although intervention strategies lag behind those for ambient air. At that time it was estimated that 1.6 million pre-mature deaths per year worldwide were caused by indoor air pollution and over half were in developing countries.

The Working Group (WHO, 2006) has taken forward work already done on ambient air quality and at a workshop in 2007, delegates agreed that many issues around ambient air pollution and health impacts were similar indoors. It recognised the complex relationship between indoor air pollution and health, the relationship between indoor air quality and building design, micro-environments that are influenced by building materials, maintenance and use of homes, and particularly the impact of indoor combustion processes and systems of ventilation. Much of the work has been done in connection with homes but these impacts will also apply equally to indoor workplaces, such as offices and banks where the indoor environment is not impacted by industrial processes.

The working group identified 3 key areas for consideration and 3 sub-groups developed the framework for new guidelines to be published.

1 Guidelines for specific agents/substances – list of compounds.

2 Biological agents – a list was produced which includes dampness and mould, ventilation systems and allergens.

3 Combustion of fuels indoors- appliances, ventilation, combustion processes and cleaner fuel sources.

From these 3 key areas it easy to discern that EHOs can have a huge role to play in developing and implementing any strategy and intervention based on these 3 key areas. EHOs already have statutory powers that include maintaining and improving housing conditions, reducing dampness and mould growth, improving ventilation, preventing ill health from indoor combustion sources like carbon monoxide both in the home and similar interventions to ensure healthy workplaces. Following extensive review of the literature and lack of current evidence, it is not known how local government EHOs address exposures impacting on asthmatics and whether there are training and development needs to ensure appropriate interventions to reduce risks in workplaces. A survey of Scottish EHOs was undertaken in 2006, when the Royal Environmental Health Institute of Scotland (REHIS) invited members to provide information about their post-qualification training and development but this survey did not include the approach taken by EHOs in the regulatory interventions. This research will further explore the outcomes of that survey and the role and function of this cohort of EHOs in Scotland to examine whether regulatory interventions happen with respect to indoor workplace environments and impacts on asthmatics.

This research will fill the gap in the evidence base and the development of a risk management framework will be a useful tool to assist in the training and development of EHOs and will support decision making in the work place risk assessment process.

In 2005 a joint NHS Health Scotland and Health Protection Scotland report – ‘Internal Air Quality and Health’ - was circulated. This paper concentrated on the impacts from

homes as a place of shelter and warmth, and as such, assigned domestic exposures to those who spend most time there i.e. the young and the elderly. Whilst this addresses two vulnerable groups of society it did not address the risks to the working age population. The report explored behaviours in the home that were linked to the growth of micro-organisms associated with dampness e.g. mould growth, fungi and warmth encouraging the growth of house dust mites and their associated allergens, poor ventilation etc.

The report explored the inter-relationships with the ambient air pollutants as well as building materials and household occupancy and use, although did not explore this further for comparison with indoor work environments such as offices, banks that could be quite similar to the home environment, so this opportunity was missed.

The strong association to children's health further developed the theme that indoor air pollution was a significant determinant of health and draws parallels with the 'Children's Environment and Health Action Plan for Europe' (CEHAPE, 2004), which required improvements to indoor environments for children, an objective of which was 'cleaner air, cleaner lungs'.

Unfortunately, indoor workplaces were not included in the NHSHS/HPS report. The concept of multiple micro-environment exposures being a combination of home, ambient and workplace exposures for workers was omitted from the report. This relationship needs further exploration and this research will try to address some of the gaps by examining multiple exposures i.e. ambient exposures, workplace exposures and exposures in the home.

In various studies about the workplace (borhf, 2011; HSC, 2005; HSE, 2003; HSE, 2006; HSE, 2008), the only reference was to 'occupational asthma' i.e. asthma caused for *the first time* through workplace exposures such as flour and grain, latex and exposures to gluteraldehyde, isocyanates, solvents etc. There was no consideration of the impacts at work on pre-existing asthmatics for 'normal' indoor workplace environments like restaurants, offices etc.

In 2012 the Department of Health issued their 'NHS companion document' on *An outcomes Strategy for COPD and Asthma*, in which they claimed that deaths and ill health from asthma were mostly preventable provided that care was tailored to the individual and that need was met as quickly as possible. The strategy agreed that there needs to be a change in approach, from cure to prevention in order to cut the cost of treatment, improve health and quality of life for sufferers. The document suggests that there should be better collaboration between the NHS, public health officials and local authorities in connection with local air pollution, but this did not currently include indoor air quality.

At the same time NHS Primary Care Commissioning launched its *Good Practice Guide for Adults with Asthma* which provides advice for clinicians on commissioning of services and practices to improve the outcomes for asthmatic adults. The guide states that any hospital admission for adult asthma is a failure in the prevention of the condition. Whilst this statement is true, the risk management of asthmatic adults prior to hospital admission is not accounted for.

It can be seen that there are a lot of policy and strategy documents cascading from the various international committees but there still does not appear to be an effective, 'joined-up' approach at international, national and local levels to deliver holistic preventative interventions for asthmatics through regulatory bodies and health care agencies.

Many of these international and UK policy and strategy documents have not resulted in practical interventions at local level and further work is needed to bring about this change.

1.2.5 Standards for indoor air quality

The UK Committee on the Medical Effects of Air Pollution (COMEAP) produced *'Guidance on the Effects of Indoor Air Pollutants'* (2004) which recommended guideline values for indoor air for 5 parameters, NO_2, CO, formaldehyde, benzene, and benzo(a)pyrene (table 1.2.5(i) below). This guidance detailed the sources of indoor air

pollution and the potential damage to human health and identified building materials and furnishings, building design and indoor combustion processes as the key sources. However, in the study, assessment of indoor exposures relied on volunteer studies involving 'fit' adults and so it was difficult to determine the true effect of indoor exposures, especially in the case of pre-existing asthmatics or those with respiratory disorders, who need greater risk management to prevent exacerbations of the existing ill-health condition.

COMEAP considered the potential of standards for indoor air quality and concluded that this would need to include the methodology for monitoring and standards for certain parameters although no comment was made on the regulation of such standards or how regulation could be taken forward. COMEAP considered using the standards for workplace exposures or the possibility of using ambient air standards, the latter was found by the Committee be more favourable as can be seen from the list of parameters for which guideline values were suggested which are directly related to standards for ambient air quality. Workplace exposures guidance from the HSE – EH 40 (HSE, 2011(a)), details the gaseous, liquid and solid chemicals that are known to cause ill health at work, and specifically identifies compounds that are known to cause 'occupational asthma' and identifies those that can lead to sensitisation. The guidance provides information on the calculation of the 'time weighted average' (TWA) so that a calculation of the 8-hour average concentration can be determined. The 8 hour working day is used a reference period for occupational exposures in the EH 40 guidance and therefore the average hourly exposure concentration can be calculated to provide the TWA.

For example the TWA would be calculated using the method described in EH 40 as follows:-

Sum of the 'concentration x time (in hours)' for each exposure in an 8 hour shift and then divided by 8 to give the hourly time weighted average e.g.

[(5ppb x 1) + (8ppb x 2) + (7ppbx 2) + (8ppb x 3)] / 8 ppb = 7.375ppb TWA.

The consideration of ambient air quality as a potential impact on occupational exposure seemed plausible as occupational exposures are based on an 8 hour working day, and as already discussed; ambient air quality is assessed continuously in time and has an impact on air quality indoors. The comparison between real-time continuous air monitoring and hourly TWAs should be reviewed cautiously as the impacts on respiratory health can be acute if short-term high concentration exposures are experienced (Bylin et al, 1988). The assessment and impact of high-concentration episodes could easily be missed when averaging concentrations over time.

Ambient air quality measurements do not reflect the impacts of multiple exposures experienced by individuals and so therefore the relationship between ambient air quality measurements and multiple exposure assessment needs further exploration. This research aims to assess and evaluate ambient air quality measurements in Guernsey in comparison with typical multiple exposures of individuals in Guernsey.

COMEAP (COMEAP, 2004) considered the possible sources of indoor pollutants and identified NO_2, CO , polycyclic aromatic hydrocarbons (PAHs) from heating fuels, formaldehyde and volatile organic compounds (VOCs) from building materials, linoleum, fabrics, carpets and floor tiles etc. particles associated with MDF fibre board, Environmental Tobacco Smoke (ETS) and a potential range of sources associated with household cleaning materials.

As yet these standards have not been implemented in legislation, which will be needed if the standards are implemented as part of national policy.

Pollutant	**Concentration**	**Averaging Time**
Nitrogen dioxide	150ppb (300ug/m^3) 20ppb (40ug/m^3)	1 hour average Annual average*
Carbon monoxide	90ppb (100g/m^3) 50ppb (60mg/m^3) 25ppm (30mg/m^3)	15 minutes 30minutes 1 hour

	10ppm (10mg/m^3)	8 hours
Formaldehyde	0.1ppm (0.1mg/m^3)	30 minutes
Benzene	1.6ppb (5ug/m^3)	Annual average
Benzo(a) pyrene	0.25ng/m^3	Annual average*

table 1.2.5(i) * provisional guidelines for indoor air quality (COMEAP, 2004)

It is interesting to note that particulates have been omitted from these provisional guidelines for indoor air quality. On reflection, this may be because larger particles are likely to fall to ground in the environment close to where they have been emitted and are less likely to be airborne over larger distances and enter buildings unless very small and light in weight (fume). The chemical composition of small air-borne particulates (PM_{10} and below) helps to determine the source of the pollution and its distribution and concentration over long distances (Qin et al, 2003). The spatial extent of the distance/concentration gradient varies by pollutant and, according to research undertaken by Karner et al in 2010, particulate matter from vehicle emissions can travel between 100-500 metres from the roadside depending on size, with an association between ultra-fine particles (PM_1 and smaller) and NO_2 distribution.

Assessment of particulate matter should be a vital component of indoor exposure assessment and has been left out of the COMEAP provisional guidelines for indoor air quality.

Gases contribute to the environment in which they are emitted and will be more likely to travel much longer distances e.g. emissions from the coal fired power stations in Yorkshire were linked to 'acid rains' in Scandinavia during the 1970s when sulphur dioxide from sulphurous fuels was emitted into the atmosphere and combined with other gases in the atmosphere to form sulphuric acid which stripped the needles from coniferous trees. The same can be said for oxides of nitrogen. According to *Enviro News* (Autumn, 2011) the acid rains of the 1970s led to a focussed campaign to reduce NO_2 levels. Whilst trends in environmental NO_2 levels are generally downward, hotspots

are known to occur and the trend there is upward. This phenomenon has been seen in Guernsey and therefore warrants further research.

1.2.6 Nitrogen dioxide and asthma

There is a large body of evidence about many of the air pollutants that impact on asthmatics, especially particulate matter (Beverland, 2007; Asthma UK, 2013; THADER, 2004; HSE, 2008; Prescott, 2000), although exposure of asthmatics indoors to NO_2 is less well researched.

NO_2 is produced through combustion processes such as heating boilers, gas cookers, coal fires in the home, and outdoors from exhaust emissions from vehicles and through emissions from industrial processes such as power stations, foundries, waste incineration etc. It can act as an oxidant and is corrosive. During the combustion process nitric oxide (NO) is produced and then this oxidises to produce NO_2. (US EPA, 2013).

In ambient air quality monitoring programmes NO and NO_2 are often added together to give concentrations of NO_X. (AEA Energy and Environment, 2008).

In the UK many local authority areas are struggling to achieve the objectives set for NO_2 in the NAQS due to traffic management problems. In fact the EU is threatening litigation if the UK does not achieve the objectives set in the EU Air Quality Directive and EC Regulations.

In Guernsey, although the UK NAQS does not apply directly, the standards are used as a benchmark until local legislation is passed to implement local air quality controls. NO_2 is the only environmental pollutant in Guernsey that is likely to exceed NAQS ambient air quality standards (States of Guernsey, 2010) and there are three locations currently where this is happening due to traffic congestion namely; Fountain Street, Bulwer Avenue and the Bridge (see locations in section 2.2). These areas are the most highly populated on the island and therefore, exceedences in air quality objectives and standards, have the greatest impact on population health. These impacts can manifest themselves in a number of ways. For example, Fountain Street is defined as a ‘street

canyon' where the traffic route has very narrow single lanes with slight bends and with high buildings on both sites which pose a major problem for the dispersal of vehicle emissions (Zhou et al, 2007; British Colombia Ministry of Environment, 2012). Fountain Street, Bulwer Avenue and the Bridge experience high traffic flows during peak times, usually between 8-9am and 4-6pm, and the resulting higher concentrations of NO_2.

NO_2 is a gas that causes irritation of the airways often leading to mucous production. In some studies mucous production leading to infection of the respiratory tract has demonstrated an increased likelihood of asthma attack in children. (Linaker et al, 2000).

NO_2 can also cause irritation to the eyes and nose. High dose exposure can result in pulmonary oedema and diffuse lung damage (US EPA, 2013).

Studies have shown that healthy adults have little or no respiratory response to the low levels of NO_2 typically found indoors. However, asthmatics will have an increased response to allergens if exposed to NO_2 before or at the same time and children exposed to levels of NO_2 are 20% more likely to develop mucous and respiratory infections causing wheezing (Tunnicliffe et al, 1994; Chauhan et al, 2003; COMEAP, 2004).

In 1992, Hasselblad et al reported that long-term chronic exposure to levels of NO_2 in the home as low as 15ppb caused a 20% increase in the risk of respiratory disorders in infants compared with a similar less exposed cohort, although this was refuted by Samet et al (1999) who claimed that there was no correlation of such exposures and impacts on infants. Further studies by Samet (2004) have recognised a correlation with elevated concentrations of NO_2 and exacerbation of asthmatic symptoms.

Rusznak et al (1996) concluded that asthmatics exposed to elevated levels NO_2 before or during exposure to allergens enhanced and exacerbated the airway response to the inhaled allergen. NO_2 exposure to levels of 400ppb for one hour caused exacerbation of symptoms which were prolonged for up to 48 hours after exposure.

In other studies exposure to NO_2 concentrations of 500ppb for half an hour showed a significant reaction which was measured by analysis of increased release of histamines in patients. (Bylin et al, 1988; Samet et al, 2004).

Chauhan et al (2003) examined personal exposure to NO_2 and the severity of asthmatic symptoms following viral infection. 114 children were monitored over a 13 month period and it was recorded that there were significant increases in lower respiratory tract infection and asthma exacerbation with continuous exposure of NO_2 during the study period at levels from 5-20 $\mu g/m^3$ (2.6 – 10.5ppb).

In some asthmatics moderate bronchial obstruction appeared to be followed by significant increases in bronchial sensitivity and when exposed to allergens and low concentrations of NO_2 (Orehek et al, 1976).

By contrast other studies have shown there to be little exacerbation of asthma symptoms during exercise or at rest if the levels of NO_2 exposure is 250ppb measured over one hour (Jorres et al, 1998). Other studies showed concentrations of up to 600ppb had shown little airway challenge to asthmatics over short periods.(Goodman et al, 2009).

These studies need to be considered in context, many being very short term studies i.e. half an hour or an hour of exposure to NO_2 and an allergen, whereas 8 hour exposure needs to be considered in the workplace and total exposure assessment needs to underpin any risk assessment carried out for pre-existing asthmatics if symptoms are to be managed effectively and holistically. Peaks in NO_2 levels have shown correlation to exacerbation of asthmatic symptoms.

Sunyer et al (2002) reported that asthmatics had a higher risk of dying from asthmatic episodes when levels of ambient NO_2 were higher, regardless of season and that higher levels of Ozone in warmer weather had a similar effect. NO_2 is a pre-curser to atmospheric Ozone and was considered during this study. The relationship between NO_2 and Ozone is an important factor in the increasing numbers of patients with asthma and allergic disease. Shea et al (2008) reported that climate change, increasing levels

of ground level Ozone and other air pollutants such as NO_2, were contributing to, and were predicted to escalate, the rising number of asthma sufferers by the end of the twenty first century.

Sunyer et al (2002) also reported that the asthma symptoms experienced during this study were not confounded by the presence of increased allergen triggers like pollen and spores and that hourly average concentrations of NO_2 from 9 -177 ppb (42ppb 75% of the time) were recorded during the 5 year study period.

Studies undertaken by Gauderman et al (2005) across 10 communities in California, USA, indicated that the respiratory health of asthmatic children was negatively affected by ambient exposures to NO_2 associated with freeways and major roads near their places of residence. The study highlighted the importance of regulation of pollutants, including NO_2 at local level.

At the European Respiratory Society conference in Barcelona in September 2013, roadside pollution was cited as a major impact on respiratory health. Lead author of the report presented at the conference, Pieter Goeminne, stated that the study findings should encourage policy makers to make air quality a key focus of any transport policy which must consider the proximity of roads to residential areas in an attempt to reduce the impact of NO_2 and other traffic related pollutants on people living nearby.

1.2.7 Risk management and pre-existing asthmatics at work

In epidemiological studies ‘risk’ has two main facets; firstly the risk that a person may be exposed to an allergen and experience an asthma attack and secondly, the risk that the proposed interventions may prevent or influence the outcome (Farmer et al, 2004). Employers are required to assess the risks to their employees and in the case of asthmatics this will be an increasing task as the “children of the incidence era (1970-

2000) seek employment" (bohrf, 2011). It is anticipated that 1 in 10 people joining the workforce will be pre-existing asthmatics.

Risk is often defined as the likelihood that a hazard will happen and risk assessment is a process to establish what the hazards are that might cause harm or ill-health (HSE 2013). When assessing pollutants, risk assessment has four main facets: hazard identification, dose-response evaluation, exposure and 'risk characterisation'. (Hetes et al, 1991). Risk characterisation is the information link between risk assessment and risk management and is crucial for informed decision making by employers or those involved in policy and strategic development.

Risk management is then the process by which the identified risks are prevented, controlled or reduced to protect health and wellbeing over time. In this case, the risk of an asthmatic individual being exposed to an allergen and resulting in an episode of ill-health, such as an asthma attack, and how that risk can be managed to prevent such an exposure. The process of predictive risk assessment helps to identify the hazard, i.e. the exposure to allergens and pollutants, and therefore, effective management of that risk will assist in preventing that exposure (HSE, 2008, HSE, 2011(b), Sibbald et al, 1992).

The development of a risk management framework for asthmatics will, therefore, involve the assessment of the allergens and pollutants that are attributed to causing an asthmatic episode, the assessment of the concentrations and duration of exposures and measures that need to be considered to prevent, or minimise, the exposure at work, and then management over time to ensure continuity of controls (WHO, 2002). The risk of workplace exposures can be significant for asthmatics. Anees (2006) reported that asthmatics have a poor outcome if occupational exposures are not managed. That research indicated that the health outcomes of asthmatics improved within a year when the exposure was removed.

In the first instance, the initial workplace risk assessment is the responsibility of the employer, who has a 'duty of care' to employees and those entering the workplace, and this will include identification of all 'significant' risks and for establishments employing 5

or more people, a written health and safety policy and risk assessment are statutory requirements (HSE, 2011(b)).

Risk assessment of workplace health and safety compliance is undertaken routinely by EHOs in local authorities. During routine inspections of workplaces, the employers risk assessment is examined to ensure that the known hazards in that workplace are assessed and the likelihood of the hazards occurring is assessed to establish the risk. If hazards are present and there is the likelihood that the hazard will cause harm, the EHO will assess the management measures that are in place to prevent or control those risks. If there is no management of workplace risks then the EHO has statutory power to enforce improvements within the terms of the Health and Safety at Work etc. Act 1974 and sub ordinate legislation. It is essential, therefore, that EHOs authorised to undertake such duties and make decisions about interventions, are competent to do so. This competence requires a combination of underpinning academic knowledge of the subject, professional experience and practice in the field before such a risk assessment can be carried out.

The Health and Safety Executive/Local Authority Enforcement Liaison Committee (HELA) '*Advice to Local Authorities on Intervention Programmes and an Inspection Rating System'* is guidance that sets out the criteria for a risk scoring scheme used by HSE inspectors and local authority EHOs/authorised officers and outlines the priority risk rating system, which determines the frequency at which workplaces should be inspected. The higher the risk of activities in a workplace, the more frequent the inspections will take place. The guidance outlines the rating elements which include safety hazard, health hazard, safety risk, health risk, welfare, public risk and confidence in management. These elements are scored, using the guidance, by the inspecting officer following the inspection of the premises and discussions with the proprietor and/or staff. The extent to which impacts from indoor environments on pre-existing asthmatics is assessed by inspecting officials, is unknown at this stage and this research will aim to examine how officials undertake the risk assessment of asthmatics in the workplaces they visit and inspect.

In 2000 the Health and Safety Commission (HSC) set out its strategy to reduce the incidence of new cases of workplace ill-health by 30% by 2010. The strategy document: '*A Strategy for Workplace Health and Safety in Great Britain to 2010 and beyond'*: included the Disease Reduction Programme (DRP). This initiative focussed on Asbestos, Dermatitis and Asthma. HSE inspectors and local authority staff undertook joint training to increase an understanding of these topics and embarked on 'themed' inspections of workplaces in their respective jurisdictions with the aim of targeting the risk management of asbestos, dermatitis and asthma. The validity of targeting resources to themed, topic-based inspections has not yet been evaluated in the context of wider health and safety at work, and the improvements expected in the workplace to the health of asthmatics from this intervention is unknown.

"The 'human cost' of asthma remains unacceptably high. The economic impact of asthma is considerable. People with asthma, their health care professionals, and the health service need to work together to reduce the high human and economic burden of asthma". (Neville et al, 2003).

1.3 The Hypothesis

Having reviewed the evidence from the body of literature about the impacts of NO_2 on pre-existing asthmatics at work, the following hypotheses were formed :-

- Asthma in the UK is on the increase and air pollution is a major contributor to exacerbation of symptoms.
- Air pollution exacerbates symptoms in asthmatics and there is a co-effect if asthmatics are exposed to NO_2 before or during exposure to allergens.
- Human exposure to multiple micro-environments, particularly those indoors, is an important factor when considering risk management of asthmatics in the workplace.
- Ambient air quality is used as a proxy for human health in national policy.
- Local authority EHOs have the ability to use a wide variety of interventions to bring about improvements in health and that of asthmatics.

1.4 Aims and Objectives of the research

The aims of this research are:-

1. To evaluate the impact of air pollution on the health of asthmatics at work so that a novel risk management framework can be developed for use by employees, employers and regulators.

2. To examine whether ambient air quality is a good proxy for impacts on the health of asthmatics by examination of temporal and spatial variations in hospital admissions in Guernsey, for patients with asthma during 2008-2012 in comparison with ambient air quality data for NO_2.

3. To evaluate multiple exposures and how multiple exposure assessment can be used in conjunction with the ambient air quality measurements to identify spatial and temporal variations.

4. To identify whether micro-environments for NO_2 exist in buildings and whether building design and layout play a role in exposure assessment.

5. To evaluate the role and approach of Environmental Health Officers in assessing the risks to pre-existing asthmatics in the indoor workplaces within their regulatory remit.

6. To assess whether exposure to NO_2 is a necessary characteristic in risk assessment of pre-existing asthmatics in indoor workplaces.

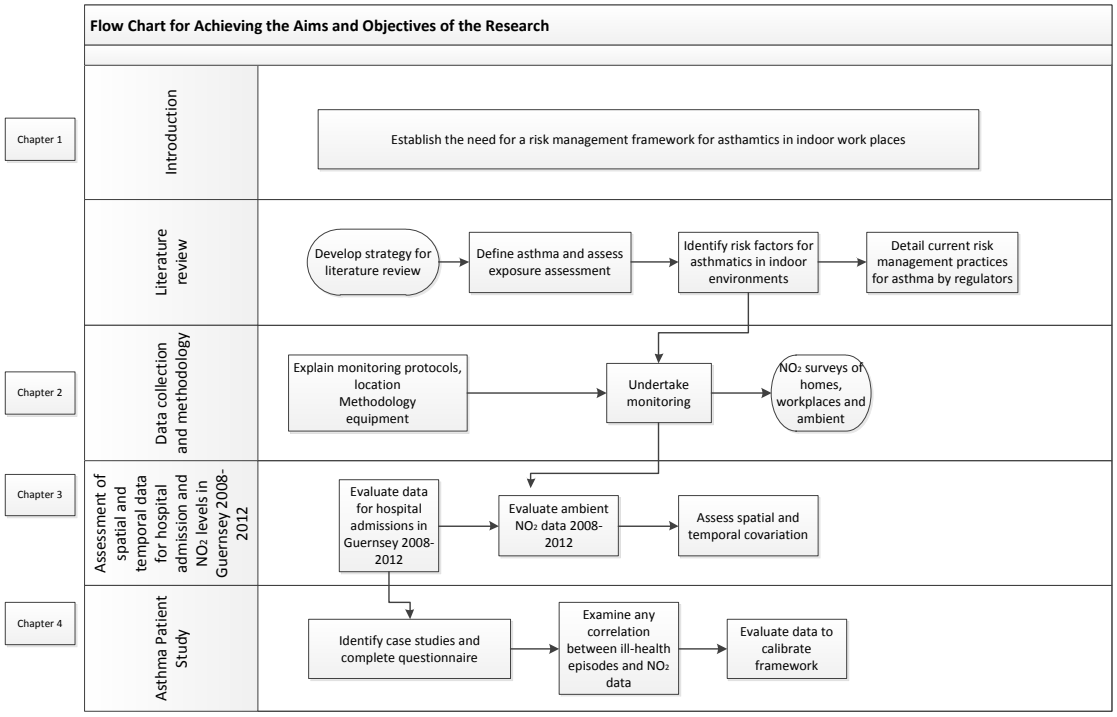
The main objectives of the research are:-

- To develop a novel framework for the risk management of asthmatics at work for easy use by employees, employers and regulators.

- To provide information about the impacts of air pollution in Guernsey, particularly NO_2, on the health of asthmatics in indoor places.
- To provide information for employees, employers and regulators about key risks to pre-existing asthmatics in the workplace.

1.5 Layout of the research

Having considered the literature and the aims and objectives of the research, the layout of the thesis was considered. A Microsoft Visio flowchart was designed that covers the scope of the studies that need to be undertaken to provided underpinning evidence for the development of a risk management frame work. This is shown as fig. 1.5(i).



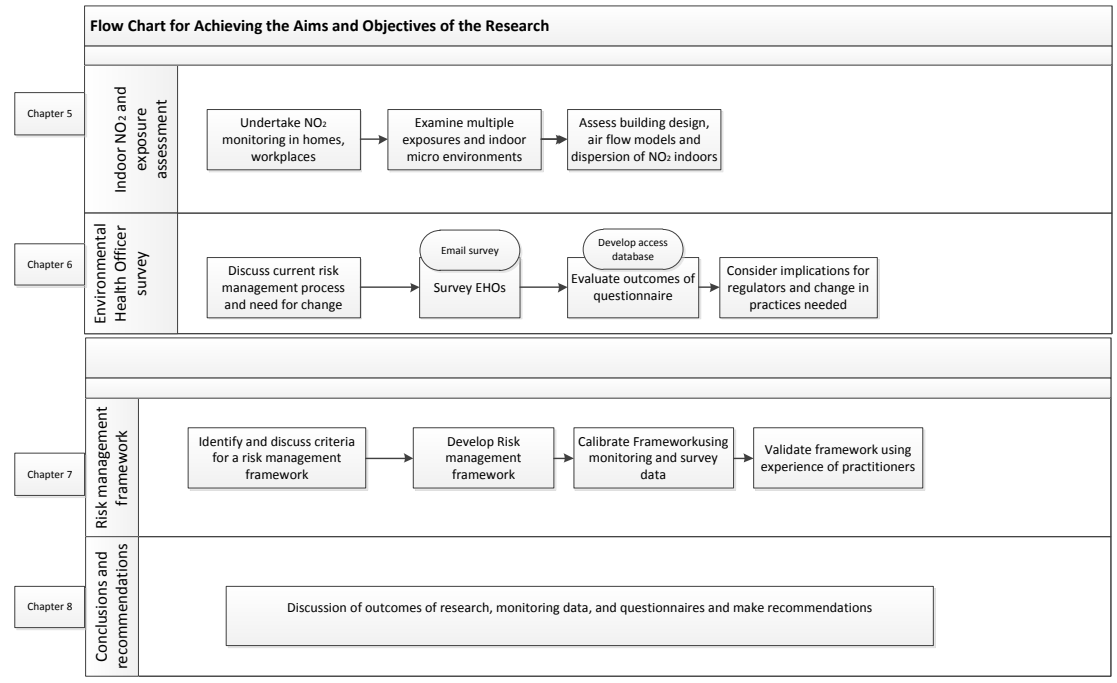


fig.1.5(i) Layout of the research

Chapter 2

Research Specification - Data Collection and Methodology

Overview

This chapter describes the data collection and methodological approach taken for this research. The objectives of the research have been determined from the review of the literature, although further details and references are included throughout this chapter.

This chapter includes a description of the study locations in Guernsey and the geographical rationale for the selection of the monitoring sites.

A number of research methods will be employed to provide qualitative and quantitative data for the underpinning evidence required to develop the risk management framework for pre-existing asthmatics at work. This chapter describes the methods used during various air quality monitoring programmes, surveys and questionnaires undertaken as part of this study.

2.1 The objectives of the research

The objective of this research is to test the hypotheses and to study a number of factors that are important to the exposure assessment and risk management of pre-existing asthmatics in indoor workplaces, as indicated by the review of the literature, and to answer the following key questions:-.

Is the current national air quality strategy, which only assesses ambient air quality, a good proxy for impacts on the health of pre-existing asthmatics indoors?

Should there be a greater emphasis on, and awareness of, exposure to NO_2 in risk assessment of pre-existing asthmatics in indoor workplaces?

Should greater importance be placed on multiple micro-environment exposures, rather the ambient air quality measurements?

Do indoor micro-environments exist for NO_2 and do they play a role in exposure assessment and is this influenced by building design indoor and air flow?

Do Environmental Health Officers adequately assess the risks to pre-existing asthmatics in the indoor workplaces within their regulatory remit?

How should the current regime of risk assessment and risk management be calibrated to ensure appropriate risk assessment of pre-existing asthmatics at work to create a simple risk management framework for employees, employers and regulators?

2.2 The Study Location

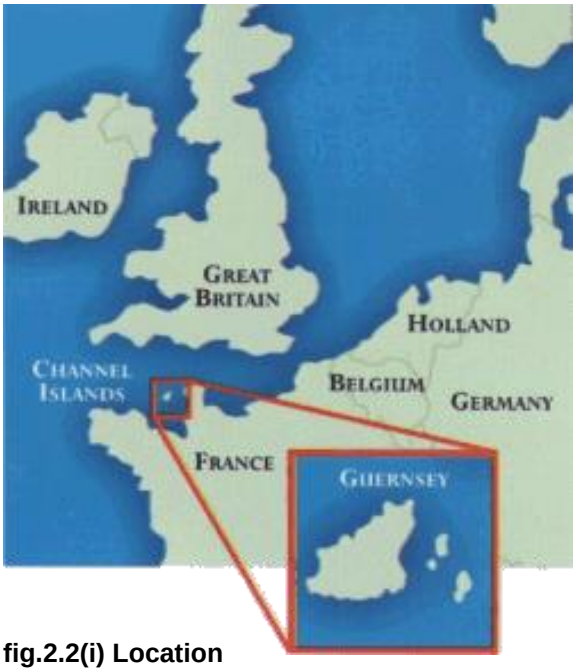


fig.2.2(i) Location

The island of Guernsey is located about 70 miles south of the south coast of England and 30 miles from the coast of France. It is bordered by the English Channel and the Atlantic Ocean and covers about 24 square miles. It is occupied by around 63,000 people. The island is divided into ten parishes and is mostly rural with the main urban centre of St Peter Port. There are two main harbours, which contribute to air pollution in St Peter Port (the town) and St Sampson (the Bridge).

Most of the commercial development focusses on St Peter Port, St Sampson and the Vale parishes. The road network connects all parishes with the town and consequently traffic flows in and out of the town are congested at peak times resulting in elevated vehicle emissions during those times. It is estimated that vehicles contribute about 29% of the total of emissions to atmosphere on the island (States of Guernsey, 2013).

fig.2.2(ii) Map of Guernsey

The island has one diesel power station, one hosptial incineration plant and a number of commercial boilers on vinery sites. The rest of the islands air pollution is contributed by domestic heating systems.

Guernsey is said to have one of the highest Gross National Products (GNP) in the world; and although the islanders are reasonably wealthy, health inequalities do exist.

Ambient air quality monitoring is undertaken by the Office of Environmental Health and Pollution Regulation (OEHPR). There has been an air quality monitoring programme on the island for around 20 years which involves the monitoring of particulate matter 10 microns and below (PM_{10}), oxides of nitrogen (NO_x including NO, and NO_2) , sulphur dioxide (SO_2), ozone (O_3) and carbon monoxide (CO). There are 3 stations where various parameters are continuously monitored and currently nine locations where monthly NO_2 diffusion tubes are located. Over the years NO_2 has been measured at over 30 locations but now the programme has been prioritised to the areas of greatest exposure (States of Guernsey, 2010). During 2013, BTX (benzene, toluene and xylene) diffusion tubes were also deployed on the island around the power station and the airport to assess hydrocarbon emissions. In 2010, the OEHPR undertook an air quality screening and assessment exercise which considered all of the objectives of the UK NAQS in the Guernsey context. The assessment revealed that the levels of the majority of parameters measured were well within the objectives for the standards set in the UK except for NO_2, where there are three locations that periodically exceed the standard, Fountain Street in St Peter Port, Bulwer Avenue and the Bridge. This research, therefore, focusses on NO_2 emissions as there has been no research into the extent of exceedences that may have an impact on population respiratory health and consequently asthmatics in Guernsey.

Standard Units

It should be noted that the monitoring equipment deployed provides data measured over various exposure periods in ppb. The NAQS provides standards in various units, some in parts per billion (ppb) and some in micro-grams per cubic metre ($\mu g/m^3$).

This study analyses data that has been measured in ppb, but where required this has been converted to $\mu g/m^3$ to allow comparison with UK standards. The conversion factor for NO_2 is 1pbb=1.91 $\mu g/m^3$ (UK Defra, 2103).

2.2.1 Ambient air quality monitoring sites and equipment

The Grange is one of the busiest roads in Guernsey being one of the 'feeder routes' from the western parishes into St Peter Port, where most people work. Real-time analysers are located here and provide data every 15 minutes on NO, NO_2, NOx and CO emissions. The equipment deployed includes an Enviro Technology (ET) 200E NO_X analyser and an ET 300E CO analyser. All are operated to ISO standards. The data is downloaded on a daily basis to a stand-alone computer in the OEHPR. For security and power supply purposes, the station is sited within the grounds of the States' Education department offices at Lukis House.

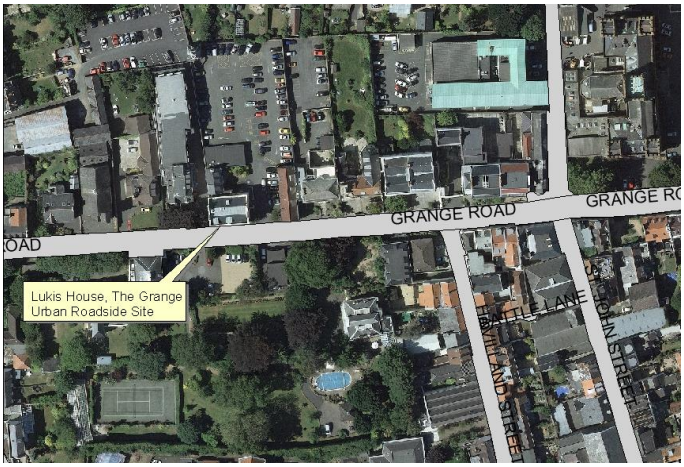


fig. 2.2.1(i) Aerial view of the Lukis House site

fig. 2.2.1(ii) Internal photo

Bulwer Avenue is another location between St Peter Port and the Bridge where there are heavy traffic flows and likely impacts from the Guernsey Electricity power station, where another real-time analyser monitoring station is located. This station is sited adjacent to the States' Environment department building. At this location the equipment includes an ET 200E NO_X analyser, and ET 200E SO_2 analyser and a Rupprecht and Patashnik Co. 1400a TEOM particulate analyser (Tapered Element Oscillating Microbalance utilising mass sensors).

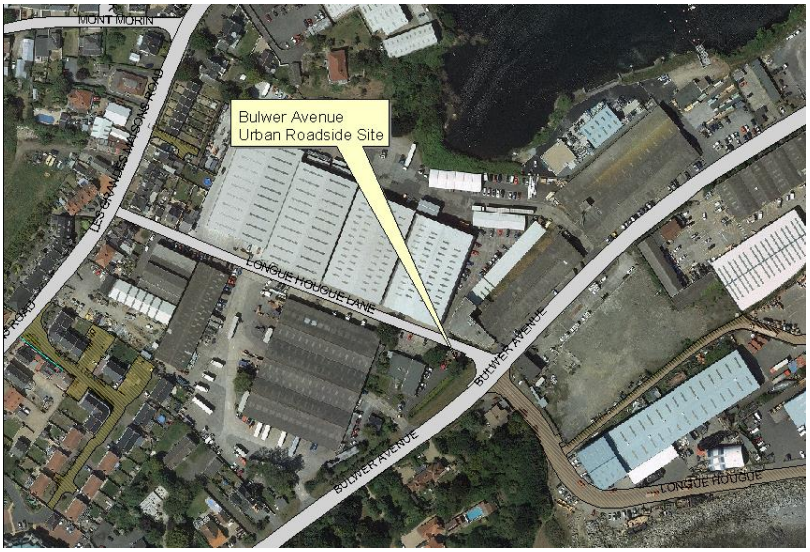


fig. 2.2.1(iii) Aerial view of the Bulwer Avenue site **fig. 2.2.1(iv) external photo**

A third real-time monitoring station is currently out of service as the site it was located at has recently been demolished and a new site is yet to be found.

Weather and Ozone monitoring is carried out at rural background site located at the dam at St Saviour's reservoir.

fig. 2.2.1(v) aerial view of St Saviour's reservoir

fig. 2.2.1(vi) monitoring site at the dam, St Saviour's reservoir

2.2.2 Diffusion tube monitoring programme.

NO_2 diffusion tubes are also located at nine sites around St Peter Port, the Bridge and the Vale.

fig. 2.2.2(i) post mounted site

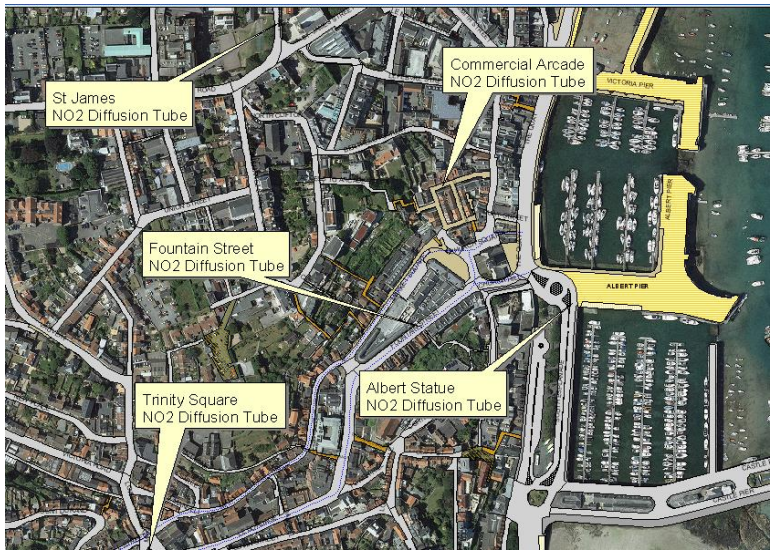


fig. 2.2.2(ii) Diffusion tube sites in St Peter Port

fig. 2.2.2(iii) Bridge -South side site

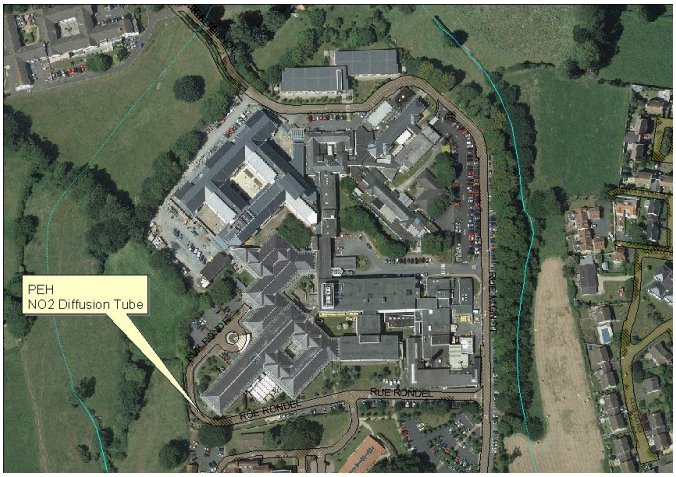


fig. 2.2.2(iv) Princess Elizabeth Hospital site

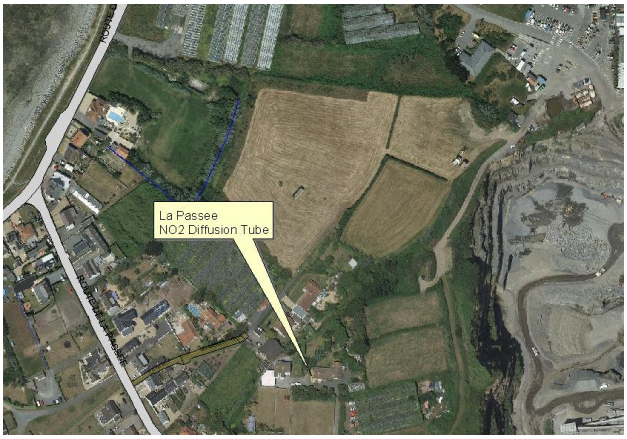


fig. 2.2.2(v) Rural site- La Passee

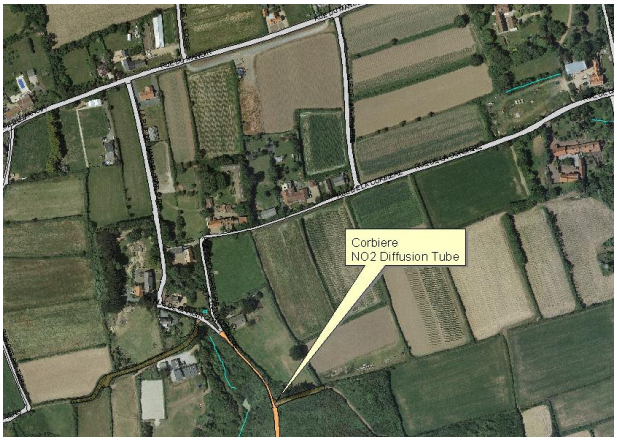


fig. 2.2.2(vi) Rural site - Corbiere

In addition to the air quality monitoring programmes for ambient air quality, a number of residences and workplaces were selected for this research so that data could be gathered to assist with exposure assessment. The residences were selected so that spacial variation in NO_2 dispersion might be measured and identified so that any impact from the various designs and layouts could be explored. Workplaces were selected to reflect the typical places of employment in Guernsey that would not usually be associated with 'occupational asthma', i.e. workplaces such as offices and food preparation. A description of each site is described in section 2.5.

2.3 Methodology

Several studies were undertaken to provide evidence for the development of a risk management framework for pre-existing asthmatics at work and to test the null hypotheses.

The studies employed real-time measurements of NO_2 whenever possible. Monthly and annual mean concentrations were assessed in line with the monitoring programmes that were developed for benchmarking against UK NAQS standards. A modelling tool for air flow was employed to estimate NO_2 exposures that may exist in indoor environments at a variety of sites.

This included :-

- a study of spatial and temporal NO_2 monthly and annual mean concentrations in Guernsey in comparison with hospital admissions data for asthmatic episodes, over the 5 years from 2008-2012 (Rosas et al, 1998; Price, 2007).
- monthly monitoring of NO_2 by diffusion tube of homes and workplaces from November 2011 to March 2012 (Price, 2007).

- the use of air circulation modelling and dispersion of NO_2 in indoor spaces using short-term real-time monitoring of NO_2 measured using a Gray Wolf gas detector to validate modelling and to determine whether micro-environments exist for NO_2. (Gray Wolf Sensing Solutions, 6, Research Drive, Shelton CT 06484, USA).
- Daily Symptom Survey of the asthma case studies (Price, 2007).
- Survey of EHOs in Scotland to assess the approach taken to risk assessment in workplaces and whether there are any training and development needs and to calibrate the risk management framework developed. (EHOs in Scotland were surveyed as this research project started in Scotland).

Throughout the research, the NO_2 monitoring studies were undertaken concurrently whenever possible to avoid temporal variation, thus reducing the need to adjust the results due to climatic condition variation.

The monitoring by diffusion tube survey was carried out from November to March to ensure that the 'worst case scenario' was assessed, based on the evidence that asthmatic episodes are exacerbated during the winter months and that higher indoor concentrations tend to be found in the winter months due to lower temperatures, damper weather conditions, closed windows, reduced ventilation etc. (Price, 2007; Wark et al, 2006).

2.3.1 NO_2 diffusion tubes

Diffusion tubes are used widely as they are relatively cheap and reliable, although can only provide an indication of mean concentrations over time for trend analysis and to give a general indication of the state of the environment in relation to the NAQS. Measurements by diffusion tube are usually taken as a monthly or annual mean, therefore, short-term temporal variations are averaged out and may be missed. Real-time analysis needs to complement this type of programme to ensure the peaks can be identified in line with standards for hourly means for various parameters, including NO_2.

Diffusion tubes used in the surveys during this research were prepared and analysed by ESG laboratories in Didcot, a UKAS accredited laboratory following ISO 17025 laboratory standards.

Diffusion tubes, or ‘Palmes-type’ diffusion tubes, consist of a small clear acrylic or polypropylene tube about 7.1cm long with two end caps. Inside one end cap there are two small stainless steel grids and this cap remains in place throughout the sampling period. The other end cap is used for security of the clean tube and is removed during deployment and exposure. It is then replaced to ensure the integrity of the sample during transport to the laboratory for analysis. During any sampling programme, the same type of tube, either acrylic or polypropylene, should be used to give consistency to the results. (AEA, 2008).

The stainless steel grids have a very fine mesh size of 4x4per mm. The grids are dipped in 50% TEA (triethanolamine) in acetone solution for at least one minute and then dried before tube assembly. This then allows for the absorption of NO_2 passing over the grids. After assembly the tubes are labelled and stored in the refrigerator before despatch. Prepared tubes will only last for up to four months so immediate use is the best practice.

Diffusion tubes are usually deployed for exposure for a month and provide an indication on NO_2 concentrations over that month. Measurements taken over time can provide an indication of concentration trends.

Siting of diffusion tubes is an important consideration. Within 1metre of a roadside there will be a direct impact from vehicle emissions and concentrations are indicative of that specific location. At sites more than 50 meters from the roadside, concentrations are more likely to reflect the general background concentrations in the area and impacts from all local sources so can be used for spatial comparisons (AEA, 2008).

The diffusion tube studies were undertaken in conjunction with the ambient monitoring programme managed by the OEHPR.

2.3.2 Diffusion Tube Siting Protocol

The sites were visited prior to the deployment of NO_2 diffusion tubes to ensure they met the following criteria (ESCAPE, 2008):-

- Houses were more than 25m from traffic flows.
- Ambient NO_2 diffusion tubes were sited away from features such as sheds, heavy vegetation.
- Diffusion tubes were located more than 100m from any large combustion process e.g. the power station, incinerator etc.
- Diffusion tubes were located to ensure there were no air restrictions around them during the sampling period.
- Diffusion tubes were mounted on brackets to ensure a minimum of 50mm off the wall/surface they were attached to.
- Diffusion tubes were located 1.5m above floor level (average respiration height) except in bedrooms where this was 1m above floor level.

One diffusion tube would be deployed in close proximity to the main combustion process in the house e.g. central heating boiler, open fire or cooking appliance.

2.3.3 Co-location studies - Gray Wolf gas detector

This equipment was borrowed from the OEHPR and so time constraints applied to its availability and use.

This hand-held gas detector was used to measure NO_2 at the same sites as the diffusion tubes to provide a number of samples for validation of diffusion tube sample results. The device was able to detect concentrations of NO_2 in ppb in real time, allowing detection of temporal peaks and variations.

The Gray Wolf gas detector was also used for rapid assessment of dispersion of NO_2 in indoor environments to evaluate whether micro environments exist. The gas detector was used to take a 2 minute sample every 10 minutes over the period of one hour at each location to identify the peak concentration observed. In addition, another study

was undertaken in house number 3 to determine whether there was spatial variation, both horizontally and vertically, in NO_2 concentrations and dispersion.

2.3.4 Changeover of diffusion tubes

The diffusion tubes were changed on a monthly basis on dates advised by the analytical laboratory and practice guidance used by the OEHPR (AEA, 2008). This coincided with the same dates as the OEHPR ambient survey so allowed comparison of short-term averaged and long-term averaged data without temporal variation.

2.3.5 Validation of NO_2 concentrations in study locations

This study was undertaken to test whether the monthly mean concentrations of NO_2 in the homes and workplaces, measured by diffusion tube, provided a realistic representation of variations in exposures to users of those indoor spaces. The Gray Wolf gas detector was used to measure NO_2 concentrations in real time. In each study location, a two minute sample was taken every ten minutes for an hour. The six sample results were then divided by six to provide an average for that hour. The rationale for the time limits for this monitoring regime was driven by access to the monitoring equipment and access to the study locations. In addition, it was considered that the sample results provided would be suitable and sufficient for this research leading to the development of a risk management framework.

This method would identify whether there were any peak variation concentrations occurring which were not identified by the diffusion tube monitoring programme, or whether the real-time concentrations were correlated with the monthly mean concentrations measured by diffusion tube.

2.4 Method 1 – Comparative study of ambient spatial and temporal NO_2 concentrations and hospital admissions for asthma in Guernsey- 2008 – 2012

- To assess whether there was any correlation between ambient NO_2 concentrations and adverse impacts on the health of asthmatics.

The methodology for this study was to manually compare data exported from the EHSCR (Electronic Health and Social Care Record system provided by InterSystems – www.intersystems.co.uk)) patient database into Microsoft Excel spreadsheets, with the data exported from the OEHPR diffusion tube survey and real time NO_2 monitoring data, also compiled on MS Excel spreadsheets (Rosas et al, 1998; Price, 2007).

The collection of patient statistical data in the Princess Elizabeth Hospital (PEH) had improved dramatically over the last few years, with the installation of a new electronic patient health information system which logged all information for patients from the admission stage, through treatment or care pathways, through to discharge from the service.

All data was coded using the International Statistical Classification of Diseases and Related Health Problems (ICD) for causes of morbidity and mortality.
The WHO publishes and distributes the ICD classification system, which is now in its tenth edition so is named ICD-10.

Asthma admissions were classified as J46 and J47 within the ICD-10 classification system, so it was these categories that were of interest to this study. When a patient was admitted to hospital the medical or clinical practitioners would keep detailed records about the patient's condition, experiences and treatment pathway. From the patient record information, clinical coders were able to add an alphanumeric code to that patient's electronic record for easy sorting, grouping and data evaluation and assessment. The clinical coders at the PEH used UK guidance on ICD-10 which details

how to undertake their coding activities and input the data onto the hospital EHSCR information system. This consistent approach allowed the data to be compared with UK data if required.

For many patients, there were a number of diagnoses made by the various clinicians involved with the case and therefore a number of alphanumeric codes were applied to the electronic record. The clinical coders in the PEH provided assistance for the data to be extracted which included the data for admissions in categories J46 and J47, which related specifically to asthma, from January 2008 to December 2012 inclusive.

The data was anonymised for this research to protect the identities of the patients, but the data was grouped to provide date of birth, gender, parish of residence, and date of admission for all patient admissions.

Data was extracted using bespoke health information software - EHSCR – and exported into Microsoft Excel spreadsheets to allow data manipulation and comparison (see Appendix 3 for the full data set used in the research).

This study was concerned with hospital admissions for asthma exacerbations that included acute or sub-acute episodes of progressively worsening shortness of breath, coughing, wheezing, and chest tightness or any combination of those symptoms. (Camargo et al, 2009).

The NO_2 air quality data was assessed for the same period. The data for the NO_2 diffusion tube surveys was extracted from the air quality data base at the OEHPR where Microsoft Excel spreadsheets were maintained. The original laboratory reports were also available for assessment and corroboration of results. In addition, real-time monitoring records were available for the period 2010-2012 and were useful in providing fifteen minute mean and hourly mean NO_2 concentrations. These data were displayed in tabular format and each day covered two pages of A4 paper. The full data set was

included in Appendix 3 on CD Rom in an attempt to save printing, although hourly measurements by month for the three years follow as line graphs in Appendix 3.

2.5 Method 2 – Measurement of NO_2 levels in indoor environments

- To establish whether there was variation between indoor and ambient levels of NO_2.

Ambient air quality monitoring data for NO_2 using diffusion tubes in Guernsey has been gathered for Method 1 and this was used in comparison with data gathered during the survey of homes and workplaces.

2.5.1 Domestic residence study locations

Three different designs of homes were selected to provide variety in location, internal layout and cooking and heating arrangements to assess and evaluate spatial variation. The NO_2 diffusion tubes were located in the homes from November 2011 to March 2012.

The layout of each house, shown as figures 2.5.1(i), 2.5.1(ii) and 2.5.1(vi), was measured and then drawn using Microsoft Paintbrush, although they were a good representation of the property the drawings were not to scale. The areas highlighted in blue were the window and door openings and the site of the diffusion tube was shown

House number 1

This was a small terraced house in St Peter Port close to Lukis House on The Grange. The property consisted of a lounge and kitchen with gas cooker on the ground floor and a bedroom and bathroom on the first floor.

NO_2 diffusion tubes were located in the kitchen, bedroom, living room and outside on the front main wall.

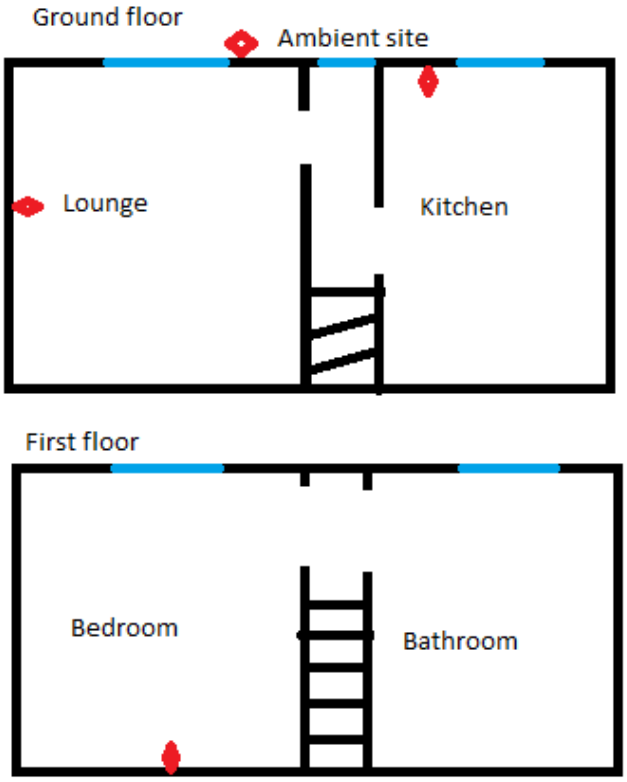


fig. 2.5.1(i) - Layout of house number 1 showing the locations of the diffusion tubes

House number 2

This was a large four bedroom house in the Vale. The house consisted of a garage with oil fired central heating boiler, kitchen dining room, lounge and bedroom/study on the ground floor and three bedrooms and two bathrooms on the first floor. The ground floor lounge had an open fireplace but this was seldom used during the sampling period. Cooking was by electric oven and hob.

NO_2 diffusion tubes were located in the garage, lounge, bedroom 1 and outside in the garden following the siting protocol. The nearest ambient diffusion tube monitoring site was Vale Avenue.

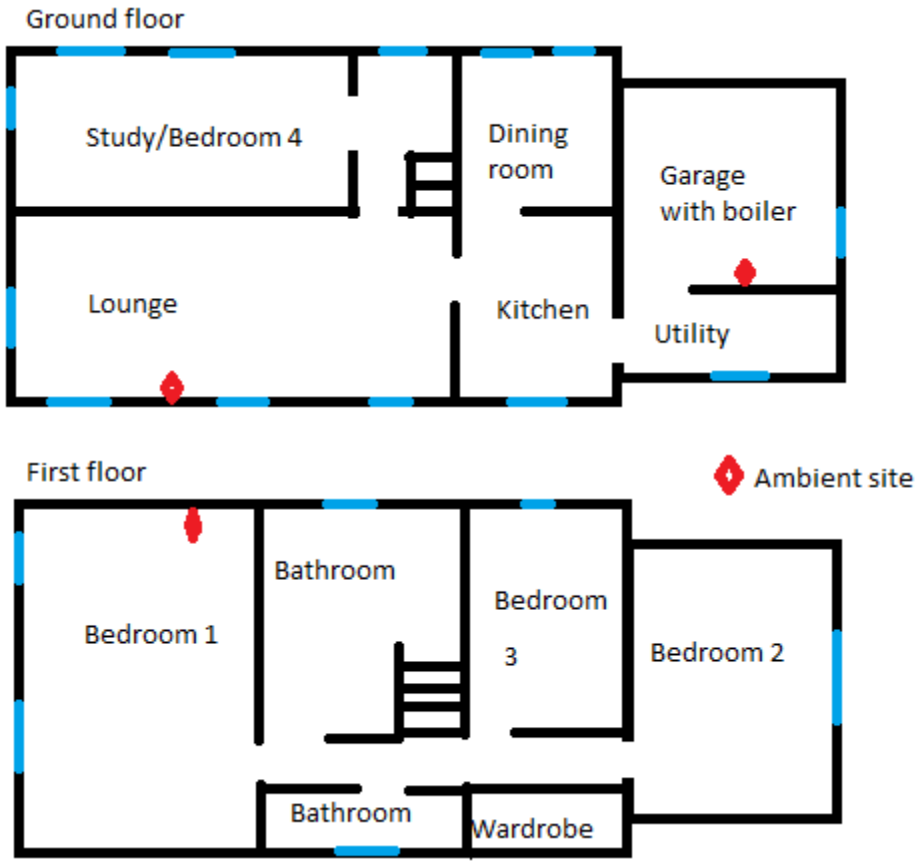


fig. 2.5.1(ii) Layout of House 2 showing the locations of the diffusion tube sites

House number 3

This was a bungalow located in Castel with two bedrooms, a gas fired central health boiler in the kitchen and an open fire in the lounge dining room. The open fire (coal and wood) was used routinely throughout the survey period. Cooking was by an electric oven and hob.

NO_2 diffusion tubes were located in the kitchen, lounge, main bedroom and outside in the garden (figures 2.5.1(iii), 2.5.1(iv) and 2.5.1(v) are photographs of the sites for house 3).

fig. 2.5.1 (iii) ambient

fig. 2.5.1(iv) Kitchen

fig. 2.5.1(v) Living room

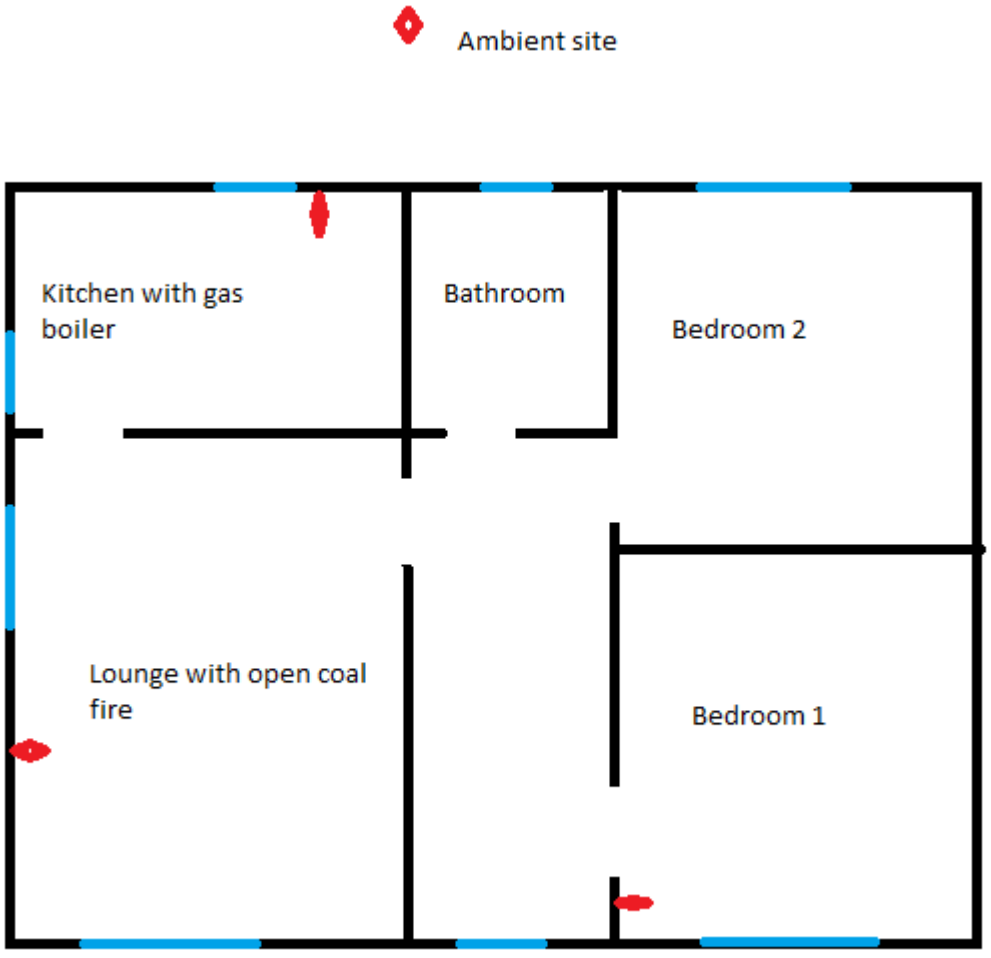


fig. 2.5.1(vi) Layout of House 3 showing the locations of the diffusion tube site

2.5.2 Workplace study locations

NO_2 diffusion tubes were located at four workplace locations from November 2011-March 2012. The sites were selected to ensure security of the diffusion tube and access for changeover. The sites were chosen because they are not typically implicated in impacts on asthmatics:-

- An office located near the main entrance to the Princess Elizabeth Hospital (PEH) which overlooked the main bus stop and was over 100 meters of the PEH waste incinerator.
- An office in the centre of the PEH Corporate Head Quarters building
- The PEH reprographics print room
- PEH central Catering kitchen

The workplaces were visited prior to deployment of the NO_2 sampling equipment. Permission was sought from the employer, The States of Guernsey Health and Social Services Department, prior to the site visit and deployment of NO_2 samplers.

The criteria listed above for house site selection were adopted for the workplace sites, which were all located within the grounds of the Princess Elizabeth Hospital in Guernsey. The sites selected also offered a level of security for the diffusion tubes deployed.

2.6 Method 3 – Air flow and NO_2 dispersion modelling

- To establish the spatial dispersion of NO_2 in both indoor residential accommodation and indoor workplaces and whether micro environments exist.

The literature revealed that spatial pollution levels in indoor environments vary considerably due to building design, air flows, ventilation and the location of combustion sources (WHO, 2002, WHO 2006, Council Directive 89/106 EEC (1989)).

The houses selected for the study, as detailed above, provided a range of designs and combustion sources in various locations, to allow assessment of a range of potential indoor micro-environments.

2.6.1 Air flow modelling

One room from each house and two offices were selected and the data inputted into the air flow model - 'Flowsquare'. (Flowsquare.com – this is two-dimensional computational fluid dynamics (CFD) software). Flowsquare was freely available on the internet and provided a user friendly and accessible tool for this research, which sought to examine basic air flow in study locations. It was acknowledged that more sophisticated software was available to architects for building design purposes but this was not readily available for this study and needed significant training to use.

Data and building layout inputted into the Flowsquare software provided a two-dimensional model that resulted in a pictorial representation of the typical air flow for that room, having regard to the location of window and door openings, combustion appliances etc.

2.6.2 Validation of Air flow and NO_2 dispersion modelling

In order to assess and validate the dispersion of NO_2 in comparison with the air flow model, real-time one minute samples were taken using the Gray Wolf gas detector.

A one minute sample was taken every 300mm horizontally and vertically at heights of 300mm, 600mm, 900mm, 1200mm and 1500mm to ensure a detailed spatial comparison could be made and to identify if there was evidence of indoor micro environments for NO_2. There were 165 samples taken on each horizontal plane and this took over five hours to complete. There were five different horizontal levels measured so this took place over five evenings in February 2012. On each sampling period the same environmental conditions existed, doors and windows were closed and the open fire was in use, burning house coal, to ensure minimal spatial or temporal variation.

The data recorded were downloaded on to Microsoft Excel spreadsheets.

2.7 Method 4 – Daily Symptom Survey of case studies in relation to heath status and NO_2 exposures

- To establish whether there was an exposure/response relationship for NO_2 levels and whether there was an impact on the health status of pre-existing asthmatics.

The data for ambient NO_2 levels in Guernsey for 2008-2012 were assessed in comparison with hospital admissions data for the same period.

Ambient NO_2 levels were measured at 12 sites across the island by monthly diffusion tube survey and real-time analysers.

Hospital admissions data for asthma were evaluated for the same period, taking account of personal patient information including parish of residence, gender, age, date of admission.

Five case studies were selected to take part in the study to run concurrently with the various house and workplace studies. The survey questionnaire was approved at the start of the research programme by the Abertay University Ethics Committee and is included at Appendix 2. The questionnaire methodology used in this research is outlined in section 2.8 and 4.2 below.

The questionnaire aimed to evaluate the health status of each case study during the months of the house and workplace monitoring programmes.

Unfortunately the first cohort of cases studies selected did not return the questionnaires and so the survey was invalid.

A further cohort of case studies was selected following the advice of the Respiratory Nurse Consultant in Guernsey. This method required additional ethics approval from the States of Guernsey Health and Social Services Department Ethics Committee, which

added to the delay in this part of the study. The ethics approved ‘participant information letter’, ‘consent form’ and ‘participant survey form’, were included at Appendix 4.

2.8 Method 5 - Survey of Scottish EHOs in relation to risk assessment of pre-existing asthmatics in the workplace

- To assess the approach taken by EHOs when to undertaking risk assessment and risk management of pre-existing asthmatics at work.

During the review of the literature it was evident that there was little information about the role and function of EHOs, whether they were engaged in national or local health and safety policy on risk assessment of asthmatics and whether this influenced their regulatory interventions. The questionnaire was designed with the purpose of asking a number of questions to provide outcomes that would address the lack of evidence. The questions were short statements, each on a single issue to prevent or reduce misunderstanding (MORI, 2013).

The survey questions were designed to assess the knowledge and understanding of asthma by EHOs and authorised officers undertaking routine health and safety at work inspections of various commercial premises with their regulatory remit.

The questionnaire was designed to ensure that the questions were open and not leading (Boynton, 2004). The layout of the form allowed the participant to answer the questions by simply ticking the box adjacent to the answer they wished to give, reducing the time the questionnaire would take to complete by busy professionals (SHU, 2013, MORI, 2013).

The questionnaire was developed to ask questions that flowed from the review of the literature such as involvement in current health and safety policies and intervention programmes. The questions included topics that would have an impact on asthmatics, such as temperature, humidity (HSE, 2008) and the work activity environment such as hairdressing, bakeries, catering, offices etc., which are in the local authority

enforcement remit, to gain data on the knowledge of the participants and how they would perceive work place environments that may impact on the health of pre-existing asthmatics. It was essential that the survey questions were phrased to prevent or reduce bias (Farmer, 2004) and to allow easy extraction of data and analysis using Microsoft Access.

A detailed survey form was agreed by the Abertay University Ethics Committee at the start of the research programme.

The Society of Chief Officers of Environmental Health in Scotland (SOCOEHS) was contacted to gain support for the research and to allow contact to be made to their staff. The letter of request and reply were included at Appendix 6.

The questionnaire was sent by email to the lead EHO for health and safety regulation in each Scottish local authority. The list of lead officers had been provided by the Health and Safety Coordinating Group (HASCOG) of Scotland, who had contributed to the development of the questionnaire.

Participants were asked to answer the questions on behalf of their local authority rather than from their own personal view point, where possible. The data was ring-fenced to ensure anonymity (MORI, 2013).

2.9 Statistical significance and analysis

Statistical significance is determined as the ‘probability’ that a ‘systematic effect’ is not likely due to just ‘chance’ alone. In scientific experiments it is not possible to test the whole population so therefore a sample is tested. In order that the outcomes of the sample tested are representative of the whole population, the statistical significance is calculated. The ‘significance’ is the mathematical probability of obtaining the same results if the test was carried out many times and achieved the same result. Probability is calculated by taking the number of favourable outcomes and dividing it by the number

of possible outcomes. Probability lies between 0 and 1, where 0 means the effect will not happen and 1 where the effect will definitely happen.

In research, statistical hypothesis testing is undertaken to ensure that the outcomes of the tests are significant, or not, and the probability that an observed effect has occurred. The inferential statistical test is the 'p-value', which is the probability of observing an extreme result if there was no true effect in the samples tested (Glasgow University, 2013).

Researchers often use the 'null hypothesis', i.e. there is no difference in the mean values of samples tested, or the 'alternative hypothesis' i.e. there is a difference in the mean values of samples tested (SRUC, 2013).

Under the null hypothesis, the 'p-value' is calculated as being the probability of an observed result arising by chance and is usually below 5% or 0.05 (p=0.05). In some medical studies a value of 0.01 (1%) is used. In this research a value of 0.05 will be used (University of Colorado, 2013).

This research project will use Microsoft Excel to provide statistical calculations detailed in this section which will be presented in an Anova table and associated graphs. The following statistical parameters will be calculated.

Regression calculations are used to demonstrate the relationship, or correlation, between two variables; one independent and one dependent, e.g. the relationship between NO_2 levels and admissions to hospital would be calculated as admissions to hospital being a dependent variable and the NO_2 levels being independent.

Linear regression tests will be used to determine the significance of a regression line and the coefficient of correlation (R). R^2 will be calculated and this represents the percentage of variation e.g. percentage of hospital admissions that can be account for by the rise in NO_2.

'Significance F' is the probability associated with this correlation. It shows the probability of such a correlation occurring purely by chance.

Chapter 3

Assessment of spatial and temporal data for hospital admissions for asthma patients in comparison with NO_2 levels in Guernsey 2008-2012

Overview

This chapter discusses the results of the evaluation and assessment of the monitoring survey outlined in Method 1 described in Chapter 2. All raw data are attached at Appendix 3.

Monthly mean NO_2 levels measured in parts per billion (ppb) have been compared with hospital admissions data for a period of 5 years, 2008-2012, to reflect the relationship between ambient NO_2 levels and the health of asthmatics in Guernsey. In addition, actual admission dates have been compared with the real-time analysis data for NO_2 over the three year period 2010-2012 when data was available.

3.1 Introduction

The purpose of this chapter is to examine the aims and objectives set out in Chapter 1 with particular reference to aim number 1:-

- To examine whether ambient air quality is a good proxy for impacts on the health of asthmatics by examination of temporal and spatial variations in hospital admissions in Guernsey, for patients with asthma during 2008-2012 in comparison with ambient air quality data for NO_2.

This study aimed to assess and evaluate data to identify whether temporal or spatial variations exist in Guernsey and whether there was a correlation between hospital admissions for patients suffering exacerbated asthmatic symptoms and ambient NO_2 levels. NO_2 was specifically selected for this study because it was the only air pollutant in Guernsey that exceeded NAQS standards (States of Guernsey, 2010). All other pollutants within the NAQS were well below the standards and it was assumed that these would not have a significant impact on respiratory health in Guernsey. In addition, the literature indicated that there was an exacerbation of symptoms when asthmatics were exposed to NO_2.

The following sections discuss the NO_2 data for 2008-2012 and the hospital admissions data for the same period.

3.2 Trends in Spatial and Temporal NO_2 monitoring data

This section provides an overview of the observed NO_2 monitoring data over time at the various monitoring locations in Guernsey as described in Chapter 2, method 1. The raw data can be found in Appendix 3. It should be noted that, during the study period, the number of diffusion tube monitoring sites varied and this is reflected in the graphs.

3.2.1 Diffusion tube survey 2008

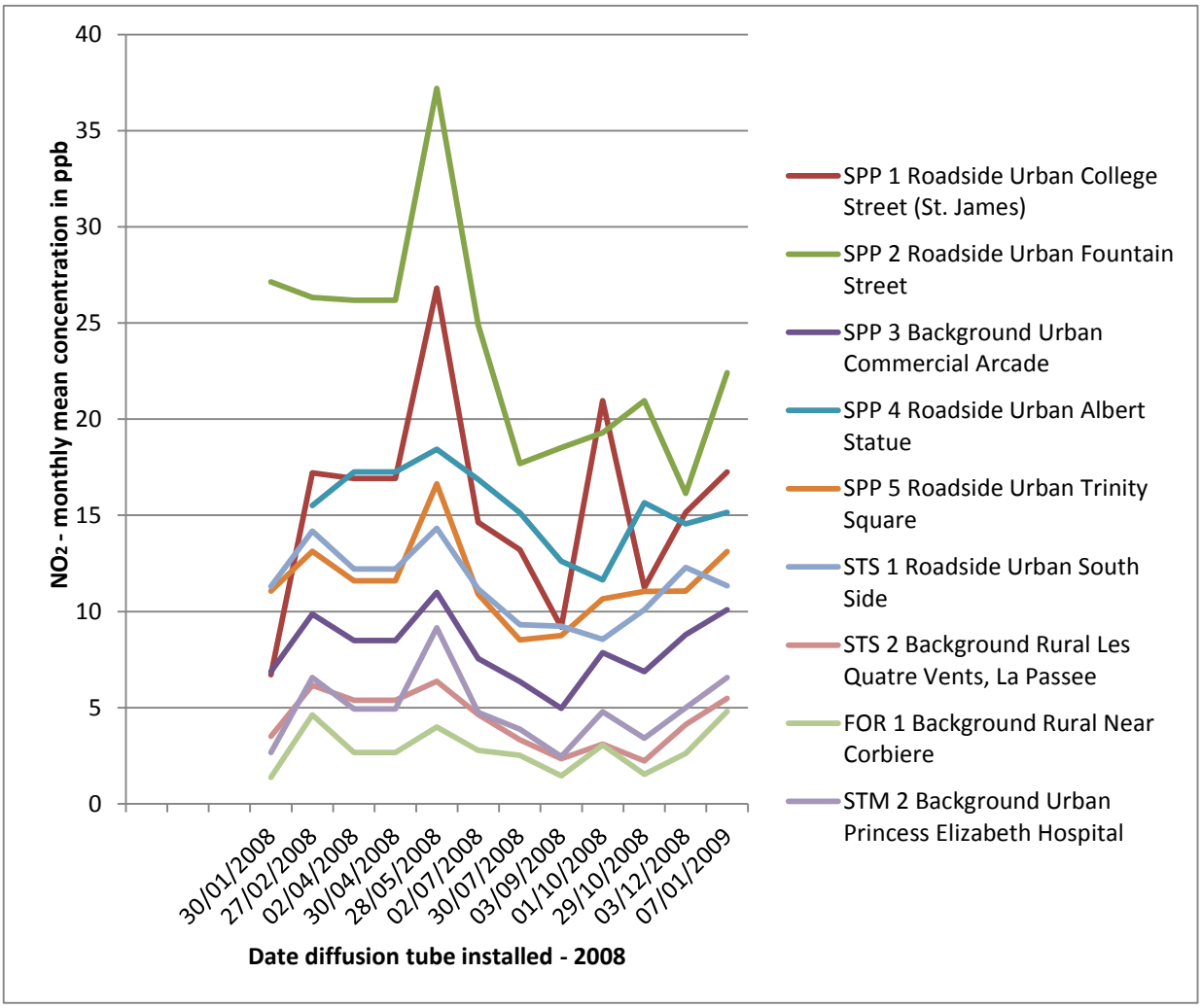


fig. 3.2.1 (i) NO_2 monitoring survey 2008 - monthly mean concentrations (ppb)

Discussion

Figure 3.2.1(i) details the NO_2 diffusion tube survey and concentrations in ppb in Guernsey for 2008 and is displayed as the monthly mean concentration by monitoring location. The legend identifies each site by colour.

From this it can be seen that the roadside urban site in Fountain Street had the highest mean concentration during June 2008 of 37ppb and this peak in concentration in June was reflected at the Princess Elizabeth Hospital site, Commercial Arcade in St Peter Port (SPP), South side, Trinity Square SPP, the Albert Statute SPP and at College Street SPP. Whilst levels dropped below 20ppb during August, possibly due to the school holidays and reduction to work related traffic jams, there was another minor peak of up to 21ppb in September, when the schools returned after the summer holidays, which is particularly prominent in College Street near Elizabeth College at the bottom of the Grange. Concentrations started to increase again during November and December 2008 rising to 23ppb in Fountain Street.

From the data, the annual mean for Fountain Street was calculated and this was 23.58ppb. Using the conversion factor of 1.91 for NO_2 this equated to an annual mean of 45.03 µg/m^3 which exceeds the NAQS standard annual mean of 40 µg/m^3.

3.2.2 Diffusion tube survey 2009

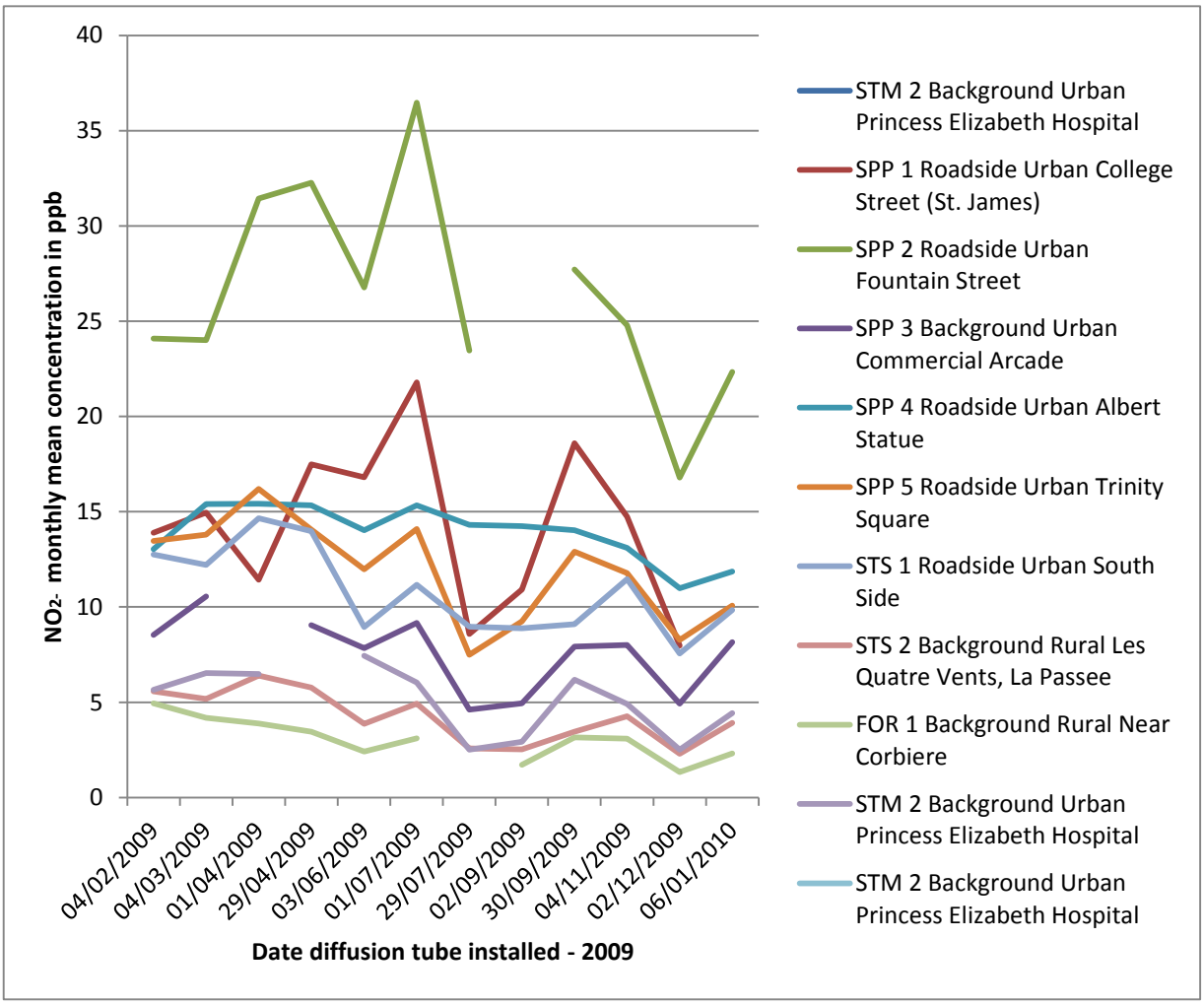


fig. 3.2.2(i) NO_2 monitoring survey 2009 - monthly mean concentrations (ppb)

Discussion

Figure 3.2.2(i) details the NO_2 diffusion tube survey and concentrations in ppb in Guernsey for 2009 and is displayed as the monthly mean concentration by monitoring location. The legend identifies each site by colour.

It should be noted that the breaks in the graph lines are due to lack of accredited data for that time period. This was due to theft of the diffusion tubes at those sites and

failures in quality assurance during transport to and from the laboratory (mainly broken tubes or displaced caps).

The peak concentration happened during June and July 2009 at all monitoring locations with a peak of 37ppb in Fountain Street. This then started to fall through August to a low level of 3pbb near the Princess Elizabeth Hospital. In September there was another smaller peak of up to 27ppb in Fountain Street and peaks at all monitoring locations which continued to fall through October and November then rose sharply again in December.

The lower concentration during August again indicated a likely correlation with the school holidays and reduction in work related traffic flows.

From the data, the annual mean for Fountain Street was calculated and this was 26.37 ppb. Using the conversion factor of 1.91 for NO_2 this equated to an annual mean of 50.37 µg/m^3 which exceeds the NAQS standard annual mean of 40 µg/m^3.

3.2.3 Diffusion tube survey 2010

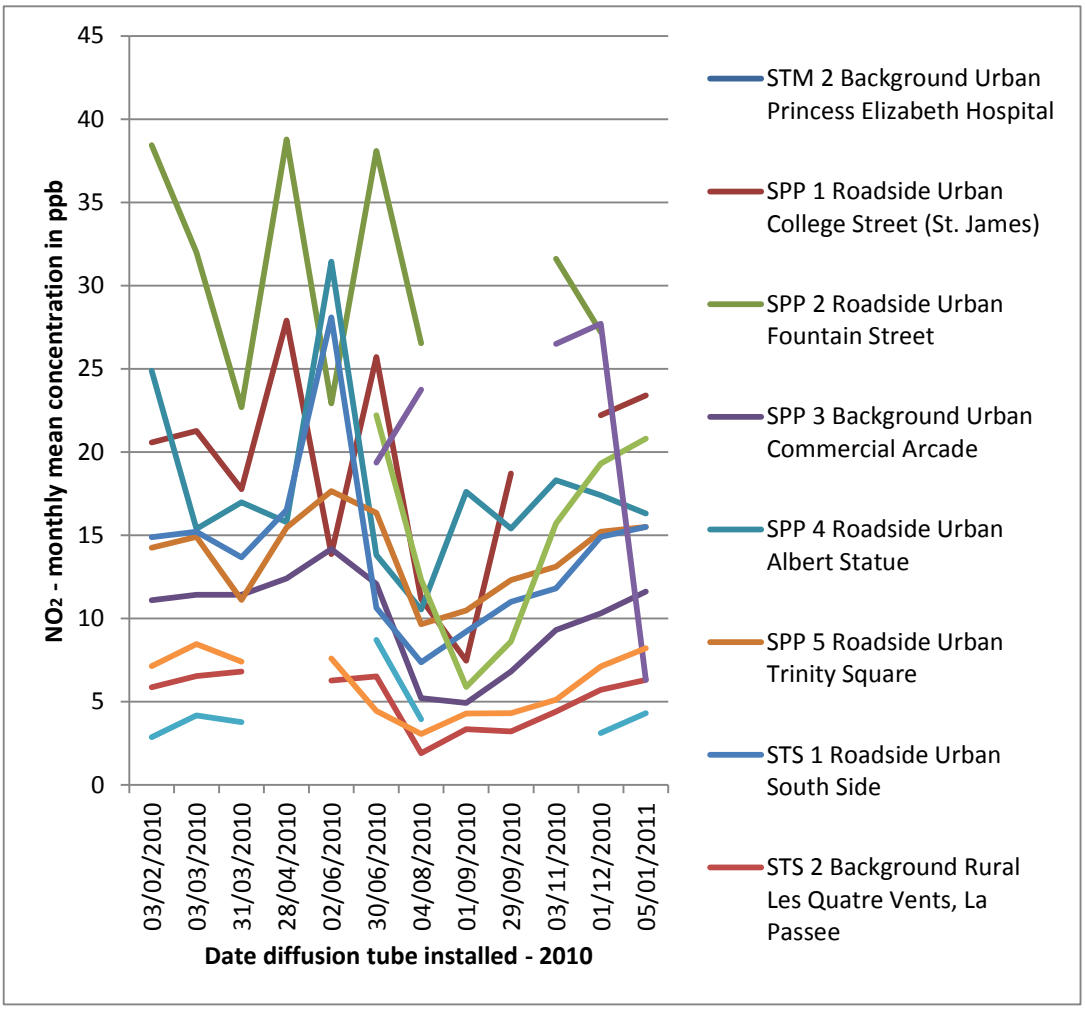


fig. 3.2.3(i) NO_2 monitoring survey 2010 - monthly mean concentrations (ppb)

Discussion

Figure 3.2.3(i) details the NO_2 diffusion tube survey concentrations in ppb in Guernsey for 2010 and is displayed as the monthly mean concentration by monitoring location. The legend identifies each site by colour.

It should be noted that the breaks in the graph lines are due to lack of accredited data for that time period. This was due to theft of the diffusion tubes at those sites and

failures in quality assurance during transport to and from the laboratory (mainly broken tubes or displaced caps).

The graph details a more erratic picture for this year. Although the peak concentrations appeared to be in June at all sites, at Fountain Street there were a number of peaks of similar concentration during February, April and June.

A similar trend appears to have developed during 2008, 2009 and 2010, in that there was a main peak in June, a fall of in concentration during August with elevation during September, fall during October and rising again during November and December 2010.

From the data, the annual mean for Fountain Street was calculated and this was 30.91ppb. Using the conversion factor of 1.91 for NO_2 this equated to an annual mean of 59.04 µg/m^3 which exceeds the NAQS standard annual mean of 40 µg/m^3.

3.2.4 Diffusion tube survey 2011

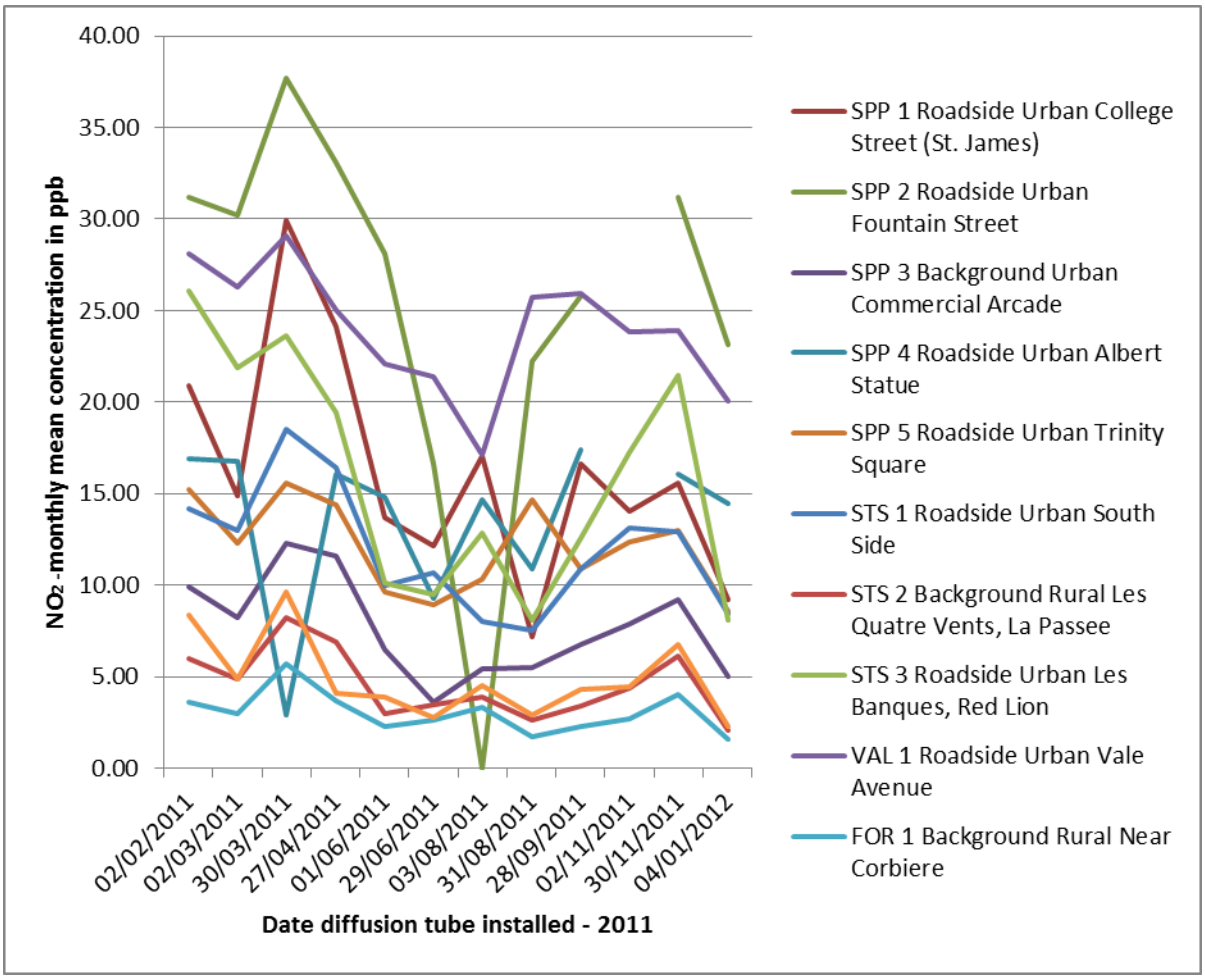


fig. 3.2.4(i) NO_2 monitoring survey 2011 - monthly mean concentrations (ppb)

Discussion

Figure 3.2.4(i) details the NO_2 diffusion tube survey concentrations in Guernsey for 2011 and is displayed as the monthly mean concentration by monitoring location. The legend identifies each site by colour.

It should be noted that the breaks in the graph lines are due to lack of accredited data for that time period. This was due to theft of the diffusion tubes at those sites and failures in quality assurance during transport to and from the laboratory (mainly broken tubes).

Interestingly in 2011 the main peak in concentration of NO_2 for all sites occurred in April, the highest being in Fountain and at 37ppb, with a general fall during July and August of 3-15ppb i.e. during the school holidays, until September when a second peak occurred at Fountain Street, Vale Avenue, College Street, Albert Statue and Trinity Square, all feeder routes into St Peter Port and prone to work and school related traffic jams.

An uncharacteristically low level could be seen in Fountain Street in August. The ESG lab was questioned about this and there appeared to have been a failure in the exposure of the tube at the location during that sampling period.

The next smaller peak occurred during November and December 2011, peaking at over 30ppb in Fountain Street.

From the data, the annual mean for Fountain Street was calculated and this was 27.92 ppb. Using the conversion factor of 1.91 for NO_2 this equated to an annual mean of 53.32 $\mu g/m^3$ which exceeds the NAQS standard annual mean of 40 $\mu g/m^3$.

3.2.5 Diffusion tube survey 2012

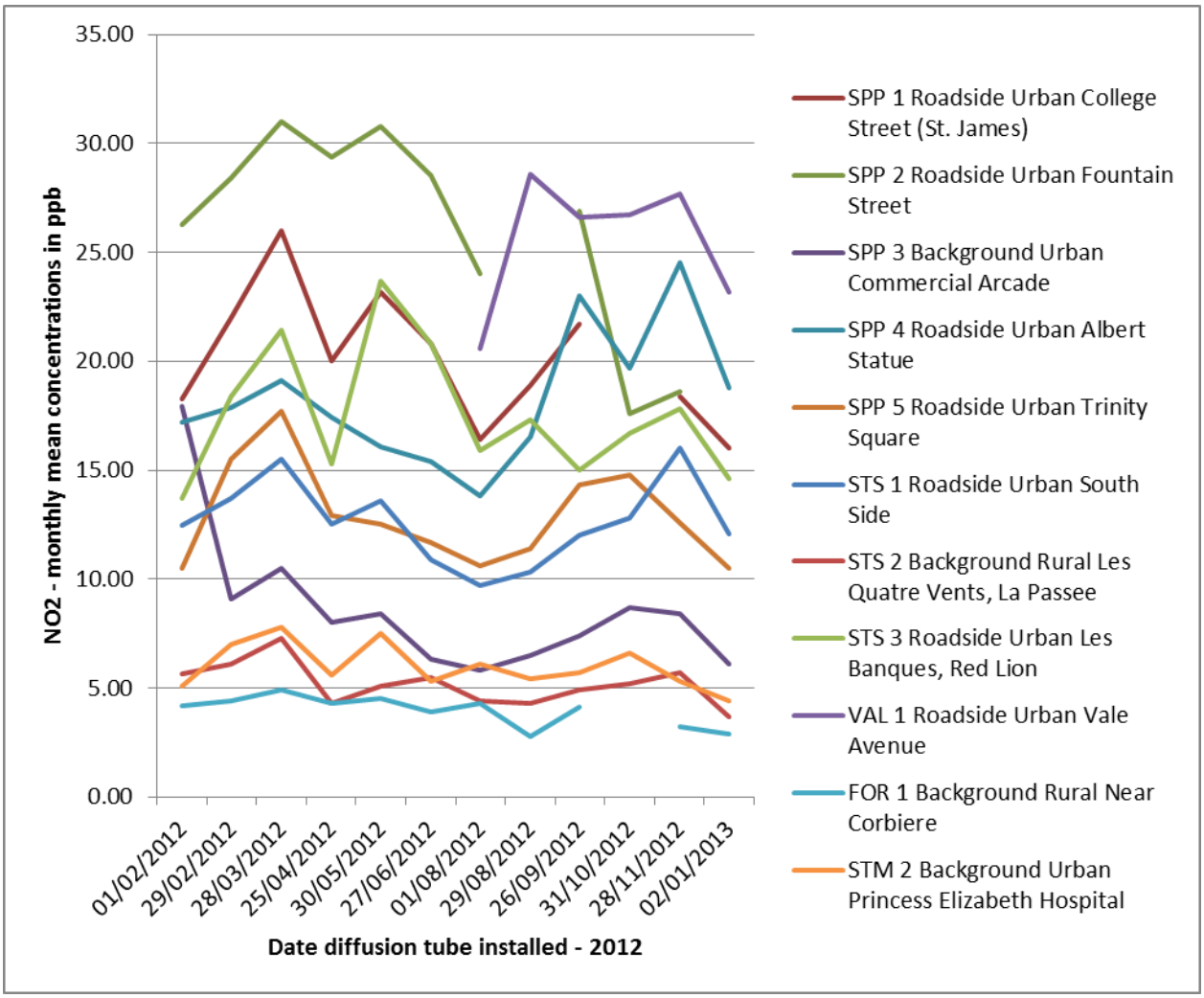


fig. 3.2.5(i) NO_2 monitoring survey 2012 - monthly mean concentrations (ppb)

Discussion

Figure 3.2.5(i) details the NO_2 diffusion tube survey concentrations in ppb in Guernsey for 2012 and is displayed as the monthly mean concentration by monitoring location. The legend identifies each site by colour.

It should be noted that the breaks in the graph lines are due to lack of accredited data for that time period. This was due to theft of the diffusion tubes at those sites and failures in quality assurance during transport to and from the laboratory.

During 2012 the main peak concentration occurred at the majority of sites in March, although the main peak concentration for Les Banques Red Lion was in May, when other sites had a smaller peak. The typical lower concentration occurred during August and then a sharp rise in September and again in November and December.

From the data, the NO_2 annual mean for Fountain Street was calculated and this was 26.15 ppb. Using the conversion factor for ppb to µg/m^3 of 1.91 for NO_2 this equated to an annual mean of 49.95 µg/m^3 which exceeds the NAQS standard annual mean of 40 µg/m^3.

3.2.6 NO_2 Diffusion tube survey – annual mean in ppb for each site 2008-2012

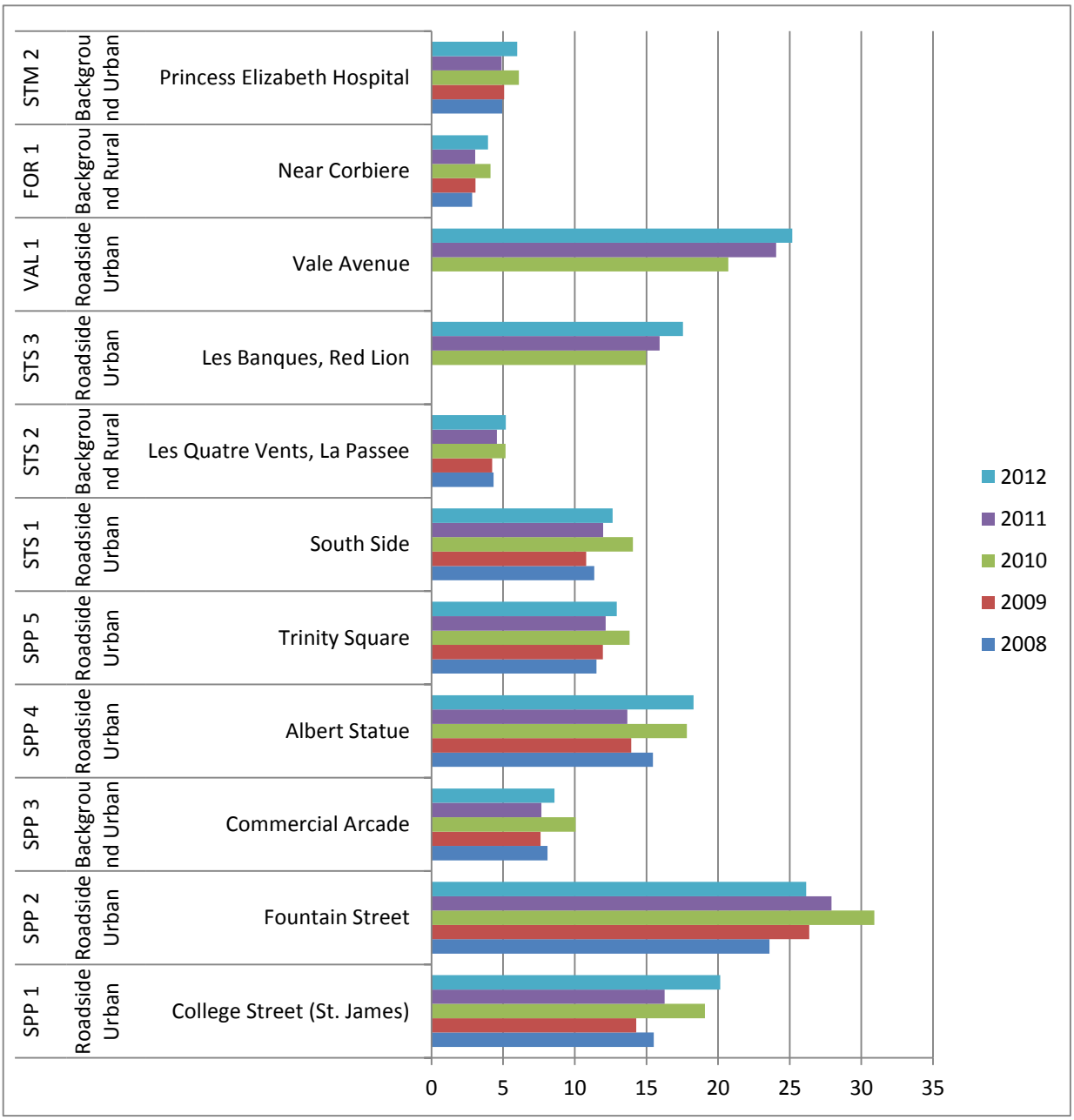


fig. 3.2.6(i) NO_2 annual mean ppb per monitoring site - 2008-2012

Discussion

Figure 3.2.6(i) shows the annual mean concentration of NO_2 in ppb for each diffusion tube monitoring site starting with 2008 as the lower bar and 2012 at the upper bar.

Several sites indicate missing data for 2011 and 2012. This is because the annual mean could not be calculated accurately due to gaps in diffusion tube monitoring data during the year.

The data show that the urban roadside site in Fountain Street was consistently the site where the highest concentrations were measured and the rural background site at Corbiere had the lowest.

NO_2 concentrations measured in the Vale Avenue site followed closely behind Fountain Street.

This clearly indicates that the areas of heavy traffic flow and therefore vehicle emissions are significant contributors to NO_2 levels in those areas where traffic jams in the morning and evening frequently occurred. Both monitoring locations were street canyon sites where the high buildings either side of the road restricted dispersion of pollutants by the wind.

For comparison purposes it should be noted that an annual mean value of 20.94 ppb equates to the NAQS annual mean for NO_2 of 40 $\mu g/m^3$. This indicates that the Fountain Street site has failed to meet the UK NAQS objective for the annual mean standard for each year from 2008-2012.

Following changes to the NO_2 diffusion survey sites in Guernsey in 2010, the Vale Avenue site was introduced and from these data it can been seen that Vale Avenue site has failed to meet the NAQS objective for the annual mean in 2011 and 2012.

3.3 Trends in Spatial and Temporal Patient Hospital Admissions

This section provides an overview of the observed patient admissions data for the Princess Elizabeth Hospital from 2008-2012. Spatial analysis of admissions for each parish was evaluated on the number of actual events per parish but the low numbers of admissions and small population makes direct comparison of data difficult. Standardised admission rates allow direct comparison geographically but due to the small numbers in the dataset, the data must be considered with care.

3.3.1 Asthma admissions to the Princess Elizabeth Hospital by month 2008-2012

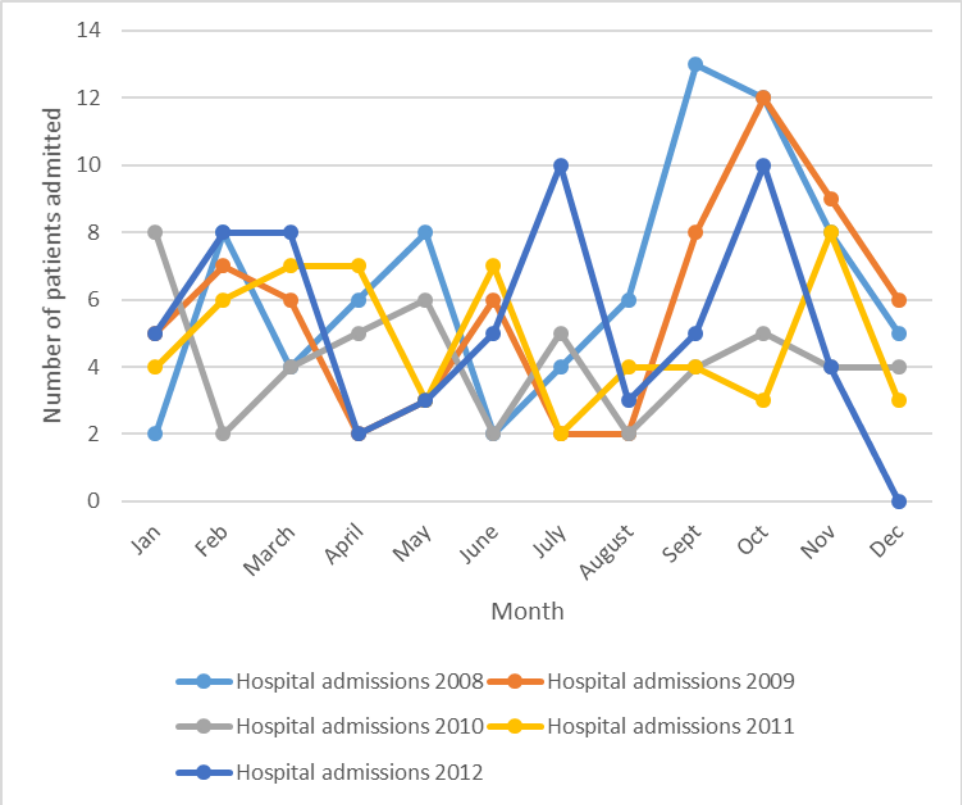


fig. 3.3.1(i) Number of hospital admissions for asthma 2008-2012

Discussion

Figure 3.3.1(i) details the actual number of asthma patients admitted to hospital each month in the years 2008 to 2012 to provide the temporal distribution of admissions starting with 2008 as the lower line graph and 2012 as the upper line graph. For each

year the distribution by month followed a similar pattern, with increases from January to February, reducing in March and April and then increasing in May. There was a steady fall during the summer months and then a steep rise in admissions in September and October, further declining in November and December. This showed that the trend across the year was consistent for the five years studied.

3.3.2 Temporal trends in hospital admissions and ambient NO_2 levels

The next part of this study is to consider hospital admissions data in comparison with ambient NO_2 levels to assess and evaluate the temporal correlation or variation.

The following graphs include both hospital admission numbers by month and monthly mean NO_2 levels measured using diffusion tubes at the ambient monitoring sites. The graphs show the data for 2008 to 2012 consecutively.

3.3.2.1 Temporal trends in hospital admissions and ambient NO_2 levels- 2008

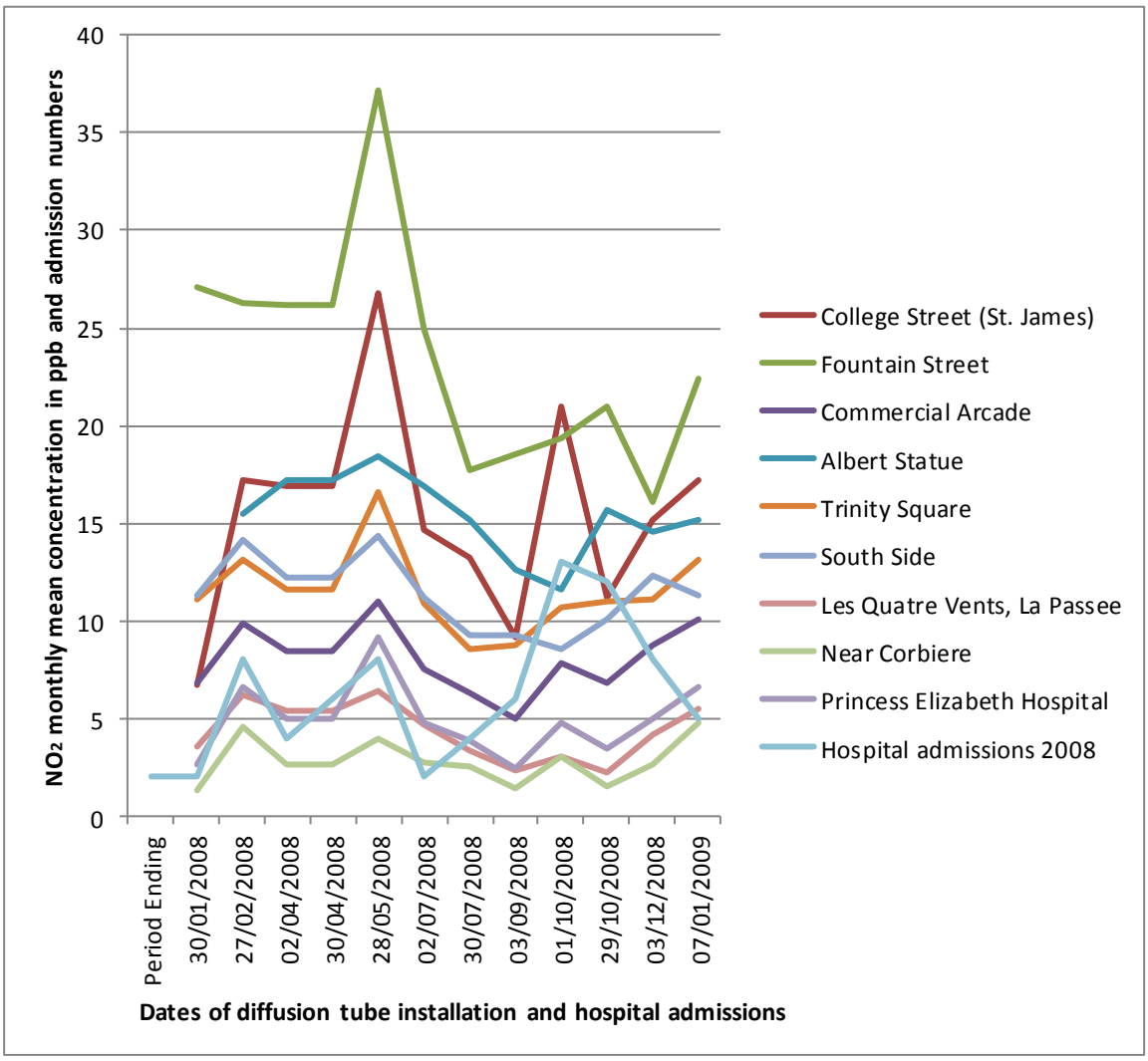


fig. 3.3.2.1 (i) Monthly mean concentrations of NO_2 and hospital admissions for 2008

Discussion

Figure 3.3.2.1(i) details the trend in monthly mean concentrations of NO_2 measured in ppb at the ambient diffusion tube monitoring sites for 2008 in comparison with monthly hospital admission numbers for 2008. Whilst the NO_2 concentrations and the number of hospital admissions were not compatible standard units, the graph details the trend. It can be seen from the graph for 2008, that when the NO_2 level rose, the number of

admissions increased and conversely when the NO_2 level fell, the number of admissions reduced. Although the trend in hospital admissions does not appear to follow any particular monitoring location, there was a general temporal correlation between ambient NO_2 levels and hospital admissions during 2008.

Statistical analysis

ANOVA	df	F	P	R2
Regression	1	0.0454	0.8355	0.0045
Residual	10			
Total	11			

table 3.3.2.1(ii) Results of regression analysis between 'mean NO_2 level (ppb)' and 'number of hospital admissions' for 2008

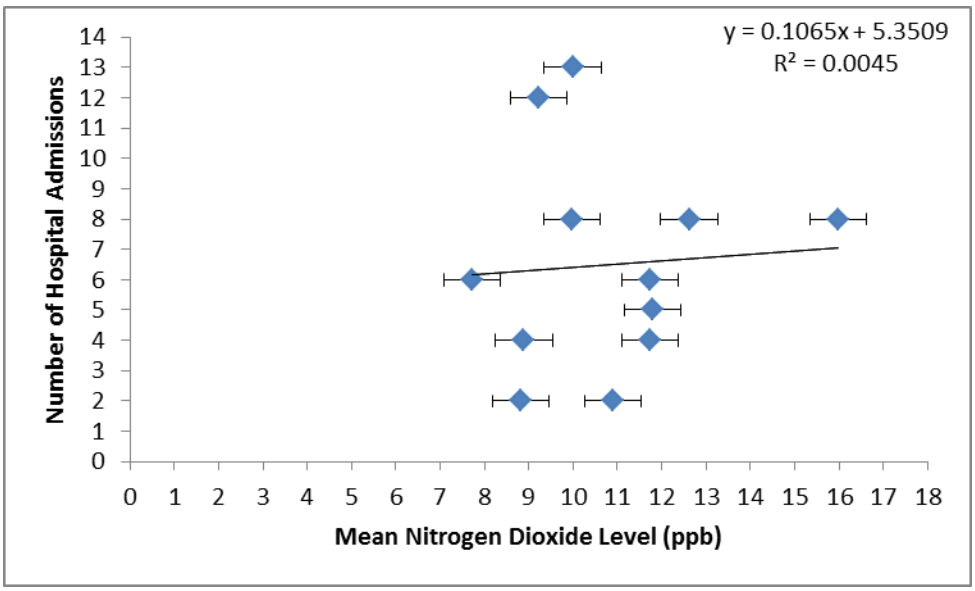


fig. 3.3.2.1(iii) Linear regression for relationship between mean NO_2 ppb and hospital admissions for 2008

The summarised results from the regression analysis from 2008 are shown in table 3.3.2.1(ii) and graphically represented in figure 3.3.2.1(iii). The R^2 value of 0.0045 indicated that only 0.45% variation in hospital admissions could be statistically accounted for by the rise in NO^2 levels during 2008.

A p-value of 0.8355 showed an insignificant result for the relationship between NO_2 levels and admissions to hospital for 2008 (95% confidence level).

The F value of 0.045 concurs that the result was insignificant.

The ‘degree of freedom’ (df) showed a total of 11.

Figure 3.3.2.1(iii) demonstrated the linear relationship between the independent and dependent variables; mean NO_2 levels and admissions to hospital respectively. Evidently there was no significant statistical correlation for 2008.

3.3.2.2 Temporal trends in hospital admissions and ambient NO_2 levels- 2009

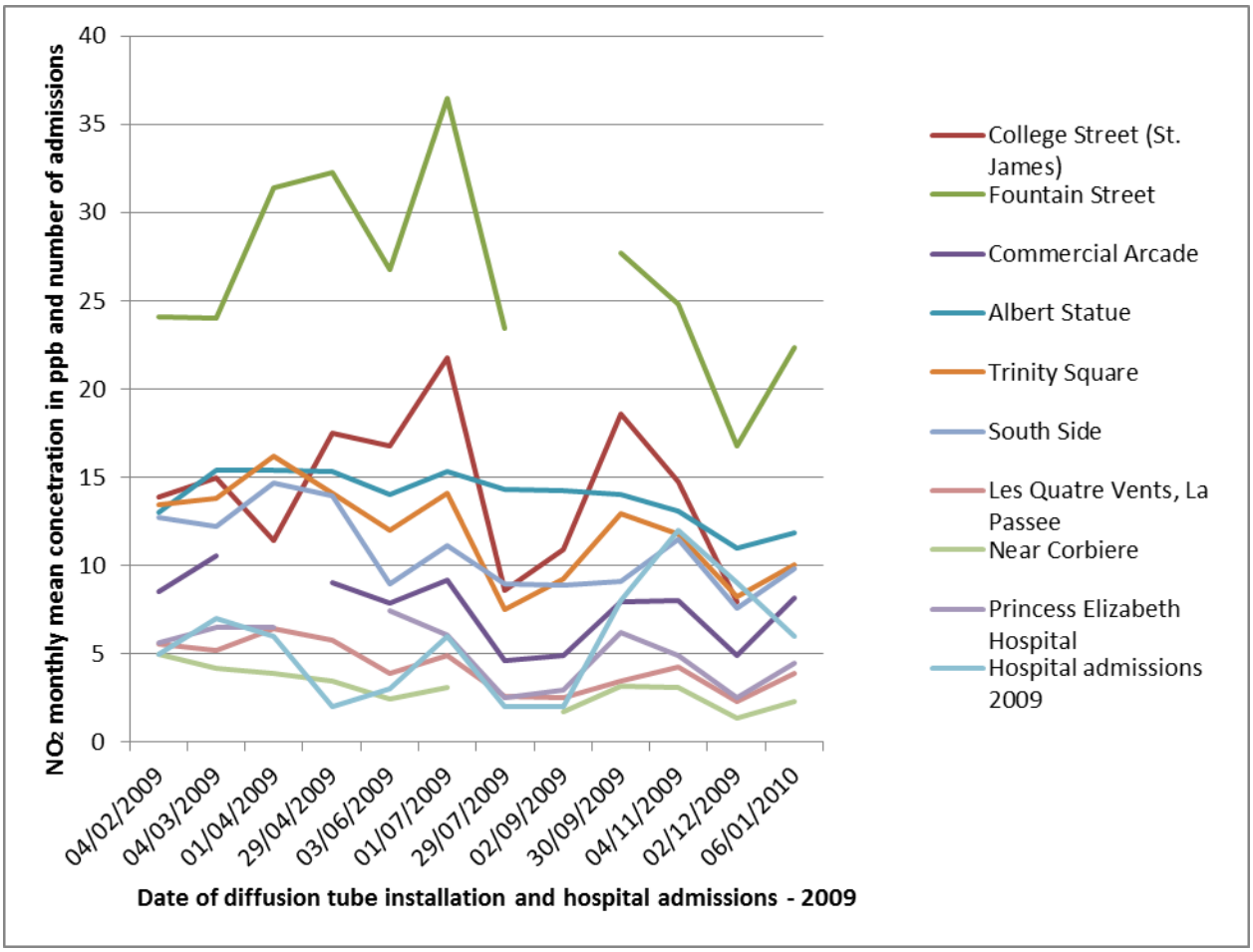


fig. 3.3.2.2 (i) Monthly mean concentrations of NO_2 and hospital admissions for 2009

Discussion

Figure 3.3.2.2 (i) details the trend in monthly mean concentrations of NO_2 measured in ppb at the ambient diffusion tube monitoring sites for 2009 in comparison with monthly hospital admission numbers for 2009. This graph is more erratic but did indicate a general trend and temporal correlation between the number of hospital admissions in 2009 and the ambient NO_2 concentrations for the majority of the monitoring locations. As the ambient NO_2 level rose, the number of hospital admissions increased and conversely as the NO_2 level fell the hospital admission numbers fell.

Statistical analysis

ANOVA	df	F	P	R2
Regression	1	0.0003	0.9873	3E-05
Residual	10			
Total	11			

table 3.3.2.2(ii) Results of regression analysis between 'mean NO_2 level (ppb)' and 'number of hospital admissions' for 2009

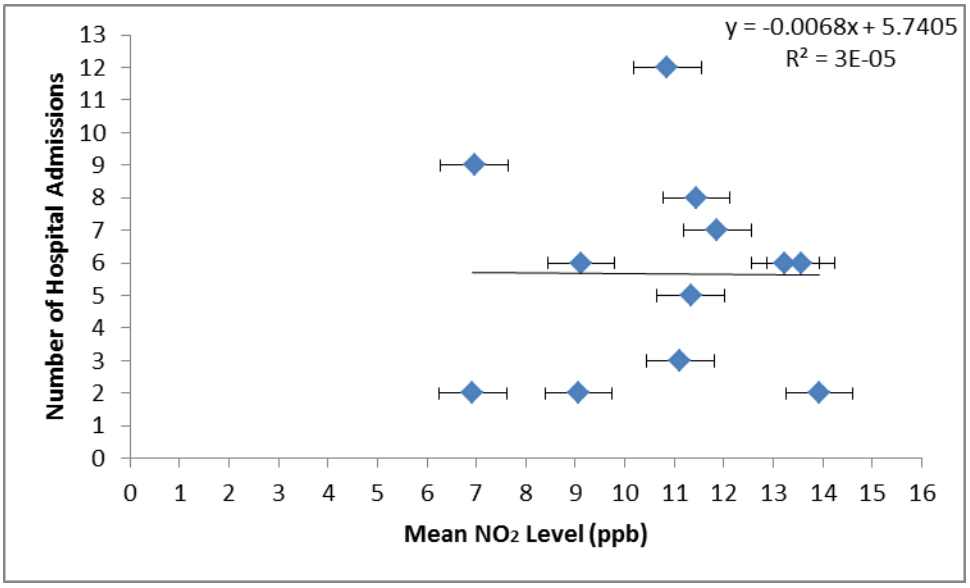


fig. 3.3.2.2(iii) Linear regression for relationship between mean NO_2 ppb and hospital admissions for 2009

The summarised results from the regression analysis from 2009 are shown in table 3.3.2.2(ii) and graphically represented in figure 3.3.2.2(iii). The R^2 value of 3E-05 indicated a minute variation in hospital admissions could be statistically accounted for by the rise in NO_2 levels during 2009.

A p-value of 0.9873 showed an insignificant result for the relationship between NO_2 levels and admissions to hospital for 2009 (95% confidence level).

The F value of 0.0003 concurs that the result was insignificant.

The 'degree of freedom' (df) showed a total of 11.

Figure 3.3.2.2(iii) demonstrated the linear relationship between the independent and dependent variables; mean NO_2 levels and admissions to hospital respectively. Evidently there was no significant statistical correlation for 2009.

3.3.2.3 Temporal trends in hospital admissions and ambient NO_2 levels- 2010

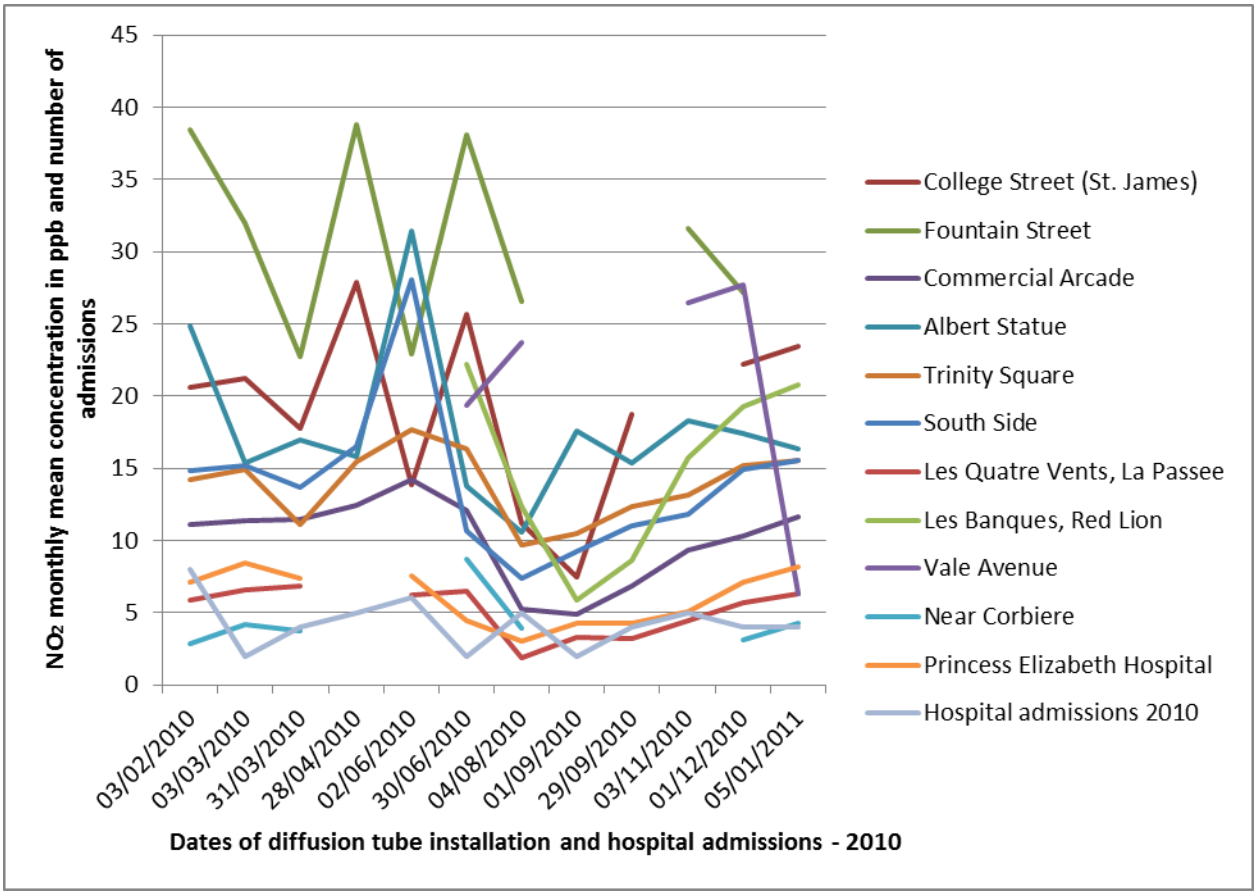


fig. 3.3.2.3 (i) Monthly mean concentrations of NO2 and hospital admissions for 2010

Discussion

Figure 3.3.2.3(i) details the trend in monthly mean concentrations of NO_2 measured in ppb at the ambient diffusion tube monitoring sites for 20010 in comparison with monthly hospital admission numbers for 2010. During 2010 the number of hospital admissions per month was low. As previously stated, care must be taken when such small numbers

are analysed. With the caveat in mind, there was a general trend and temporal correlation with the number of hospital admissions increasing as NO_2 levels rose.

Statistical analysis

ANOVA	df	F	P	R2
Regression	1	1.4189	0.2611	0.1243
Residual	10			
Total	11			

table 3.3.2.3(ii) Results of regression analysis between 'mean NO_2 level (ppb)' and 'number of hospital admissions' for 2010

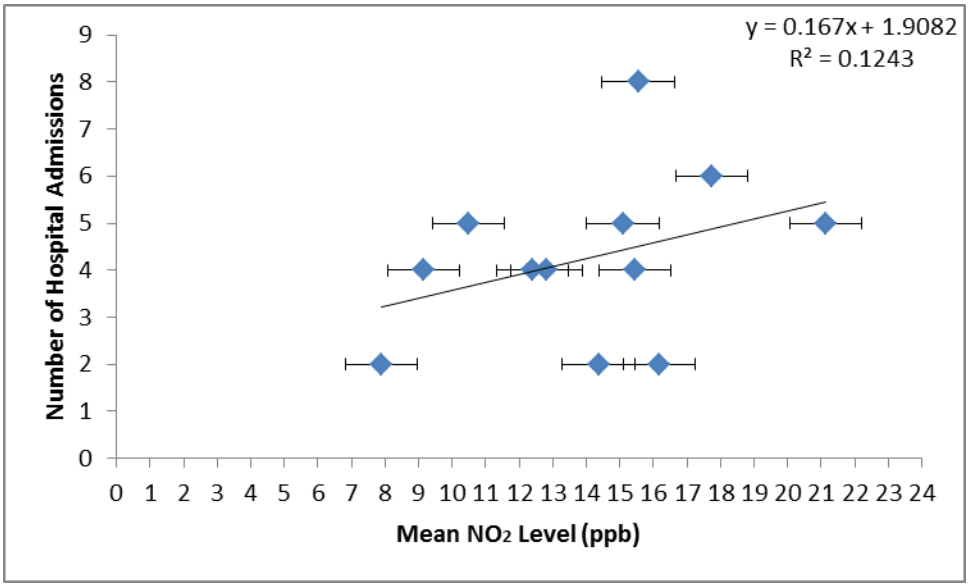


fig. 3.3.2.3(iii) Linear regression for relationship between mean NO_2 ppb and hospital admissions for 2010

The summarised results from the regression analysis from 2010 are shown in table 3.3.2.3(ii) and graphically represented in figure 3.3.2.3(iii). The R^2 value of 0.1243 indicated that only 12.43% variation in hospital admissions could be statistically accounted for by the rise in NO_2 levels during 2010.

A p-value of 0.2611 showed an insignificant result for the relationship between NO_2 levels and admissions to hospital for 2010 (95% confidence level).

The F value of 1.4189 concurs that the result was insignificant.

The ‘degree of freedom’ (df) showed a total of 11.

Figure 3.3.2.3(iii) demonstrated the linear relationship between the independent and dependent variables; mean NO_2 levels and admissions to hospital respectively. Evidently there was no significant statistical correlation for 2010.

3.3.2.4 Temporal trends in hospital admissions and ambient NO_2 levels- 2011

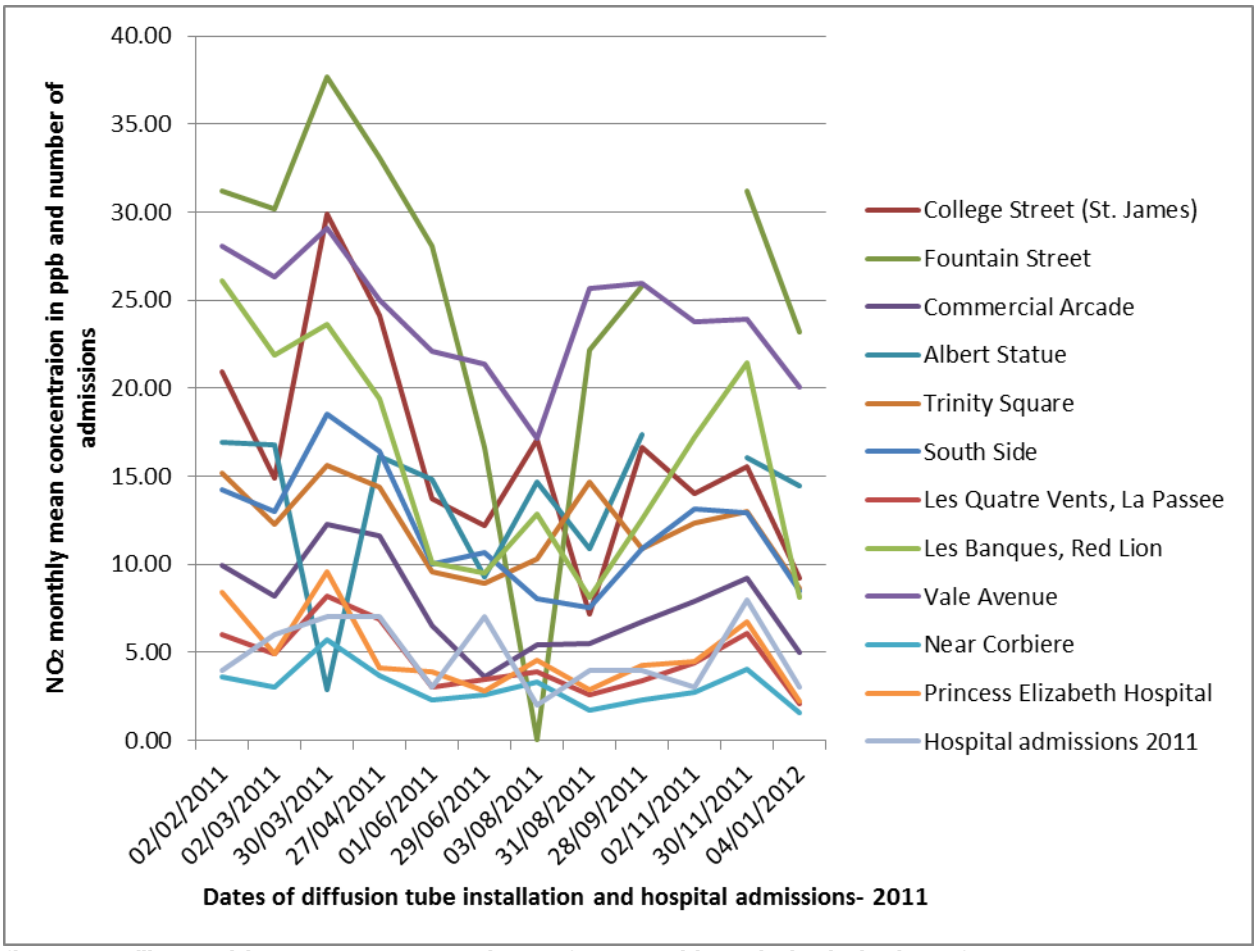


fig. 3.3.2.4(i) Monthly mean concentrations of NO_2 and hospital admissions for 2011

Discussion

Figure 3.3.2.4(i) provides details of the trend in monthly mean concentrations of NO_2 measured in ppb at the ambient diffusion tube monitoring sites for 2011 in comparison with monthly hospital admission numbers for 2011. Whilst this graph is somewhat erratic, it detailed the trend of NO_2 levels during 2011and indicated a general correlation with hospital admissions.

Statistical analysis

ANOVA	df	F	P	R2
Regression	1	4.4022	0.0623	0.3057
Residual	10			
Total	11			

table 3.3.2.4(ii) Results of regression analysis between 'mean NO_2 level (ppb)' and 'number of hospital admissions' for 2011

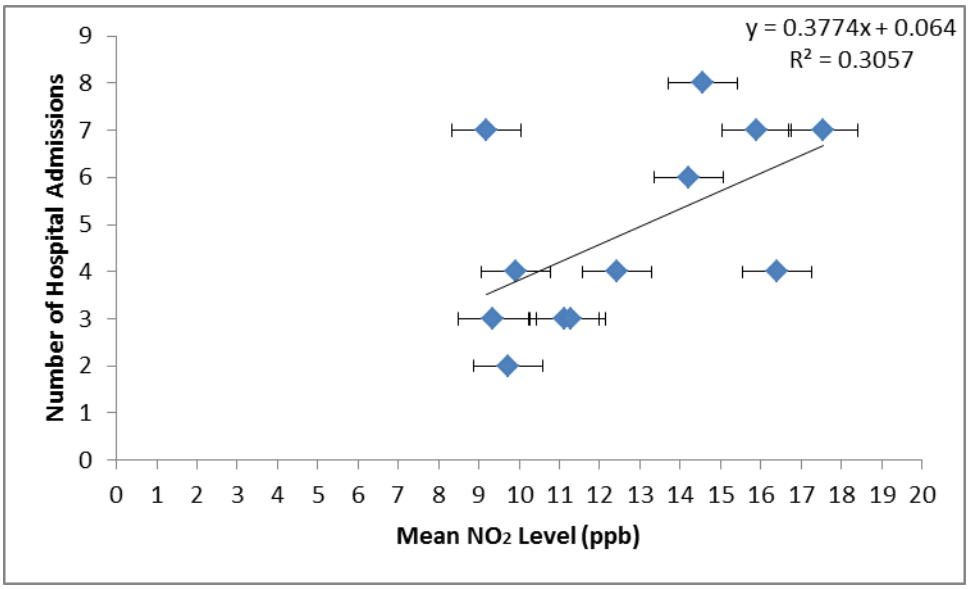


fig. 3.3.2.4(iii) Linear regression for relationship between mean NO_2 ppb and hospital admissions for 2011

The summarised results from the regression analysis from 2011 are shown in table 3.3.2.4(ii) and graphically represented in figure 3.3.2.4 (iii). The R^2 value of 0.3057 indicated that 30.57% variation in hospital admissions could be statistically accounted for by the rise in NO_2 levels during 2011.

A p-value of 0.0623 showed an insignificant result for the relationship between NO_2 levels and admissions to hospital for 2011 (95% confidence level).

The F value of 4.4022 concurs that the result was insignificant.

The 'degree of freedom' (df) showed a total of 11.

Figure 3.3.2.4(iii) demonstrated the linear relationship between the independent and dependent variables; mean NO_2 levels and admissions to hospital respectively. Evidently there was no significant statistical correlation for 2011, however, in comparison with the data for 2008-2010, the linear relationship was stronger in 2011.

3.3.2.5 Temporal trends in hospital admissions and ambient NO_2 levels- 2012

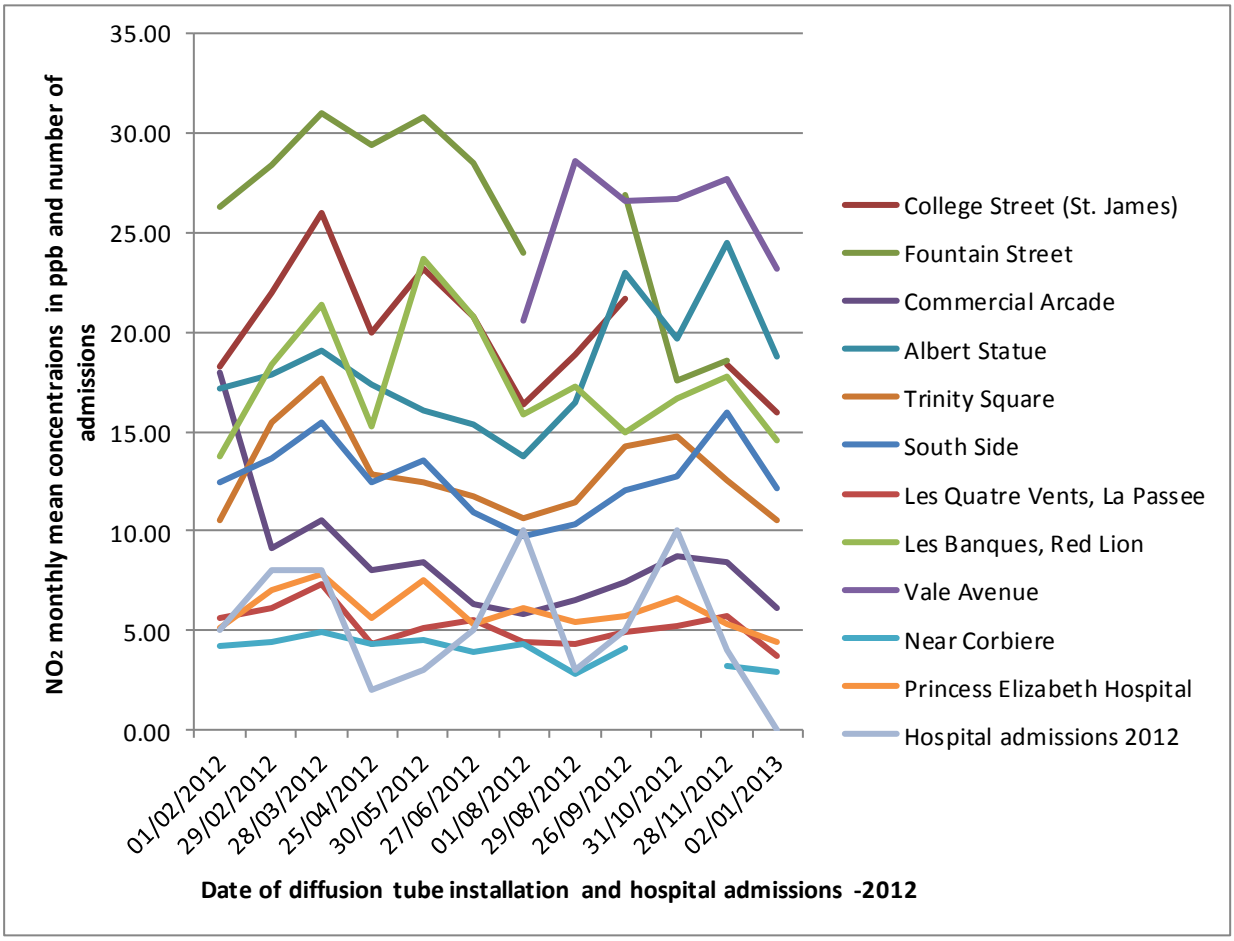


fig. 3.3.2,5 (i) Monthly mean concentrations of NO_2 and hospital admissions for 2012

Discussion

Figure 3.3.2.5(i) details the trend in monthly mean concentrations of NO_2 measured in ppb at the ambient diffusion tube monitoring sites for 2012 in comparison with monthly hospital admission numbers for 2012. There was a correlation in the trend other than for

September when hospital admissions fell although NO_2 levels peaked. At some monitoring stations the NO_2 levels started to fall in October, when the hospital admissions rose again. The lag time could have been a factor here and again the small numbers need to be analysed with caution.

Statistical analysis

ANOVA	df	F	P	R2
Regression	1	1.3973	0.2645	0.1226
Residual	10			
Total	11			

table 3.3.2.5(ii) Results of regression analysis between 'mean NO_2 level (ppb)' and 'number of hospital admissions' for 2012

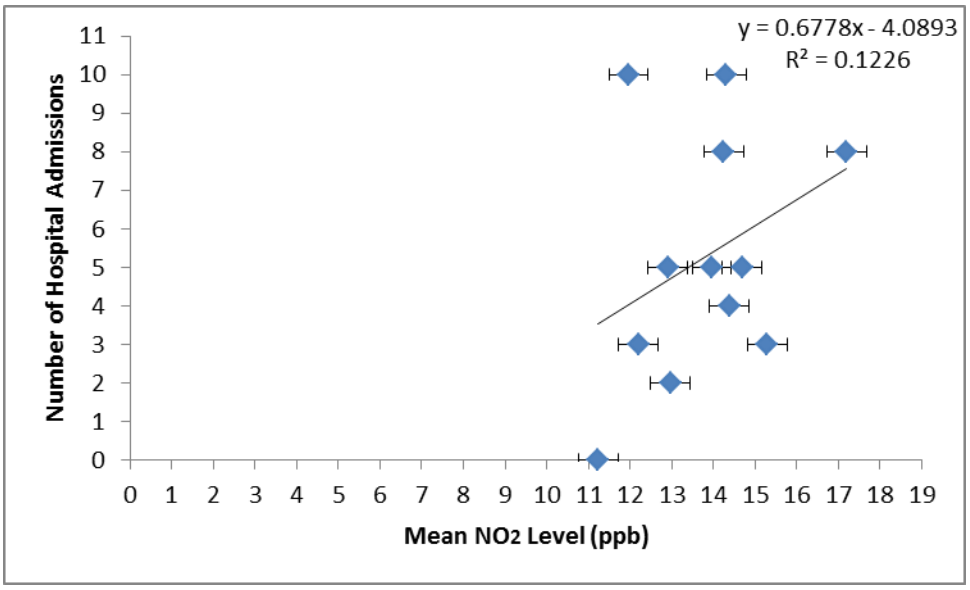


fig.3.3.2.5(iii) Linear regression for relationship between mean NO_2 ppb and hospital admissions for 2012

The summarised results from the regression analysis from 2012 are shown in table 3.3.2.5(ii) and graphically represented in figure 3.3.2.5(iii). The R^2 value of 0.1226 indicated that only 12.26% variation in hospital admissions could be statistically accounted for by the rise in NO_2 levels during 2012.

A p-value of 0.2645 showed an insignificant result for the relationship between NO_2 levels and admissions to hospital for 2012 (95% confidence level).

The F value of 1.3973 concurs that the result was insignificant.

The ‘degree of freedom’ (df) showed a total of 11.

Figure 3.3.2.5(iii) demonstrated the linear relationship between the independent and dependent variables; mean NO_2 levels and admissions to hospital respectively. Evidently there was no significant statistical correlation for 2012.

3.3.3 Spatial analysis of hospital admissions in Guernsey 2008-2012

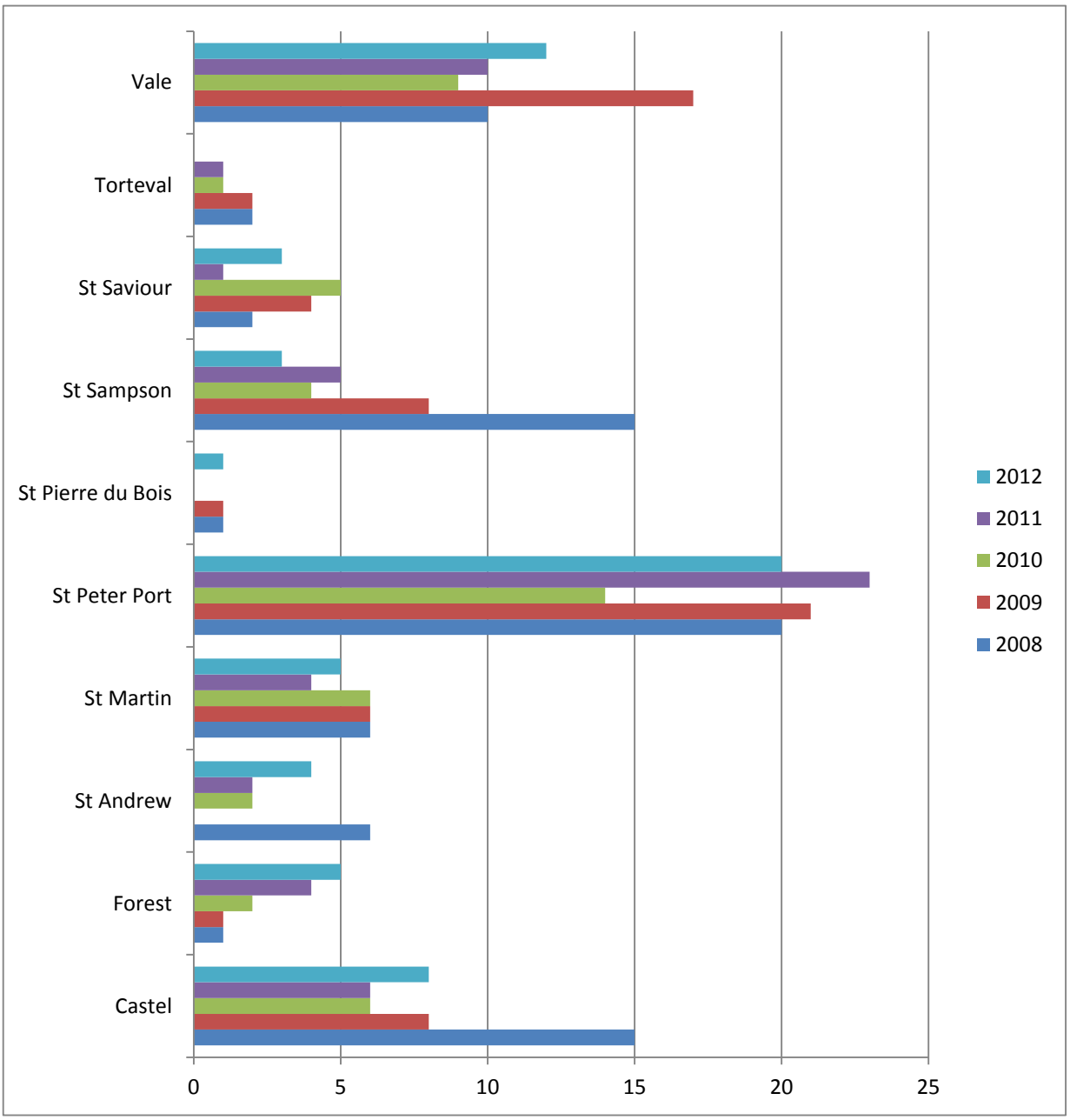


fig. 3.3.3(i) Number of asthma admissions to hospital from the parishes - 2008-2012

Figure 3.3.3(i) indicates the actual number of admissions to the PEH from 2008 to 2012 with the data grouped by parish of residence on the date of admission. The greater numbers of admissions were from the parishes of St Peter Port, Vale and St Sampson,

where the islands heaviest traffic flows occurred and consequently higher concentrations of NO_2 were recorded.

However, these parishes were also the most densely populated and therefore the admission numbers need to be standardised to allow comparison geographically. In epidemiological studies, the 'standardised admission rate' is used for this purpose. Calculation of rates can be direct or indirect and allow comparison across geographical areas, such as the parishes in Guernsey. In the UK most population health data is expressed in terms of rates per 1,000 head of population. However, in Guernsey it is very difficult to use a calculation per 1,000 population as the total population is only 63,000, with only a few thousand residents in some of the parishes (States of Guernsey, 2001). Therefore, care must be taken when evaluating such small numbers and extrapolating them into population terms (Farmer et al, 2004).

A 'population calculator' using parish population data per 10,000 was produced using Microsoft Excel (Appendix 3) so the standardised admission rate per 10,000 of the population could be determined. This gave a better indication for geographical comparison of admission rates across the ten parishes.

3.3.4 Standardised admission rate per parish per 10,000 population

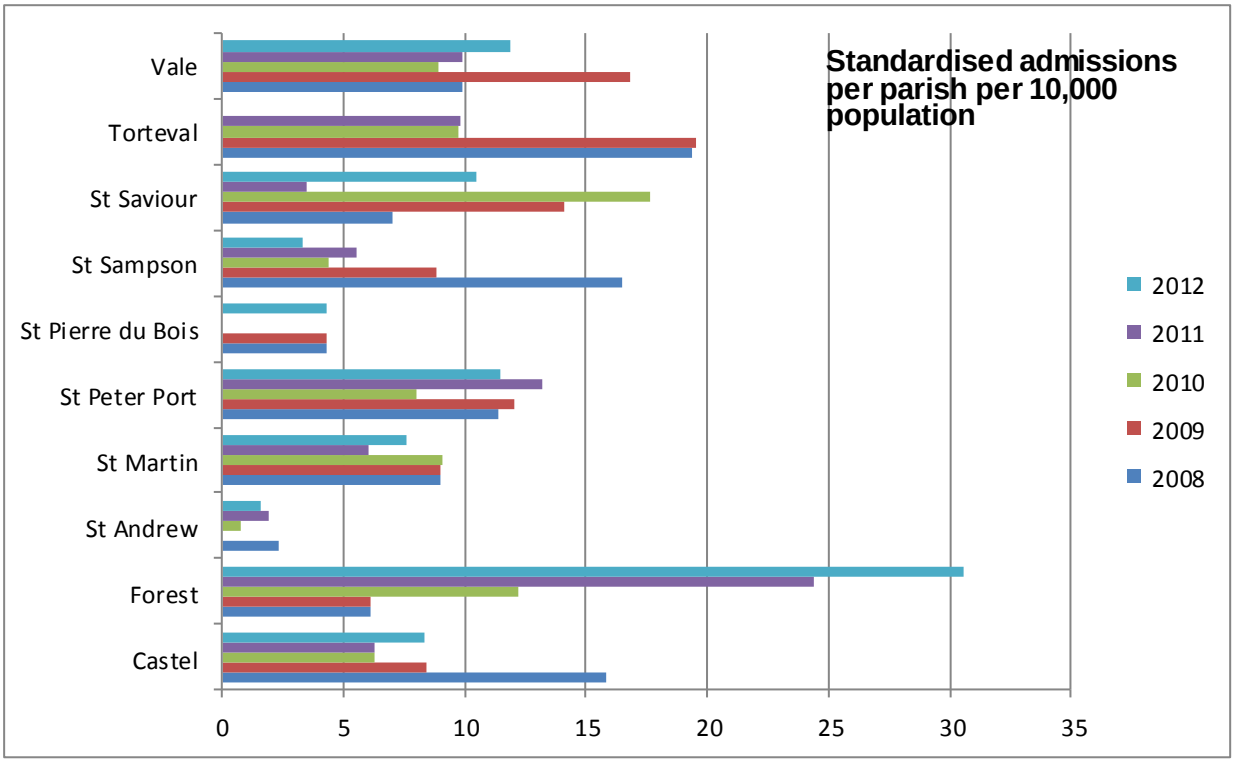


fig. 3.3.4(i) Standardised admission rates per parish

Figure 3.3.4(i) revealed a different picture to the non-standardised data in figure 3.3.3(i). Figure 3.3.4(i) indicated that the spatial distribution of hospital admitted patients was reasonably even across the ten parishes. This indicated that asthma rates were evenly distributed across the Guernsey population and were not parish specific.

Of particular interest, was the admission rate for the Forest, which showed an elevated parish admission rate during 2012. This parish was generally rural but hosted the island's only airport. During 2012 major redevelopments works were undertaken with in excess of 3000 heavy lorry movements carrying aggregates, sand and cement, from St Sampson's harbour to the airport and thousands of movements by other construction vehicles and plant at the airport site contributing to the usual aircraft movements. In addition, the works involved excavation and concrete crushing operations which caused elevated particulate levels in the immediate area and which will have contributed to the discomfort of those suffering from respiratory conditions, including asthmatics.

It was reported that one asthmatic resident was rehoused due to ill health when the development works started. The impact of the airport redevelopment works on the health of local residents in the vicinity of the airport will be worthy of further research in future after the development has been completed.

3.3.5 Demographic distribution for hospital admissions for 2008-2012

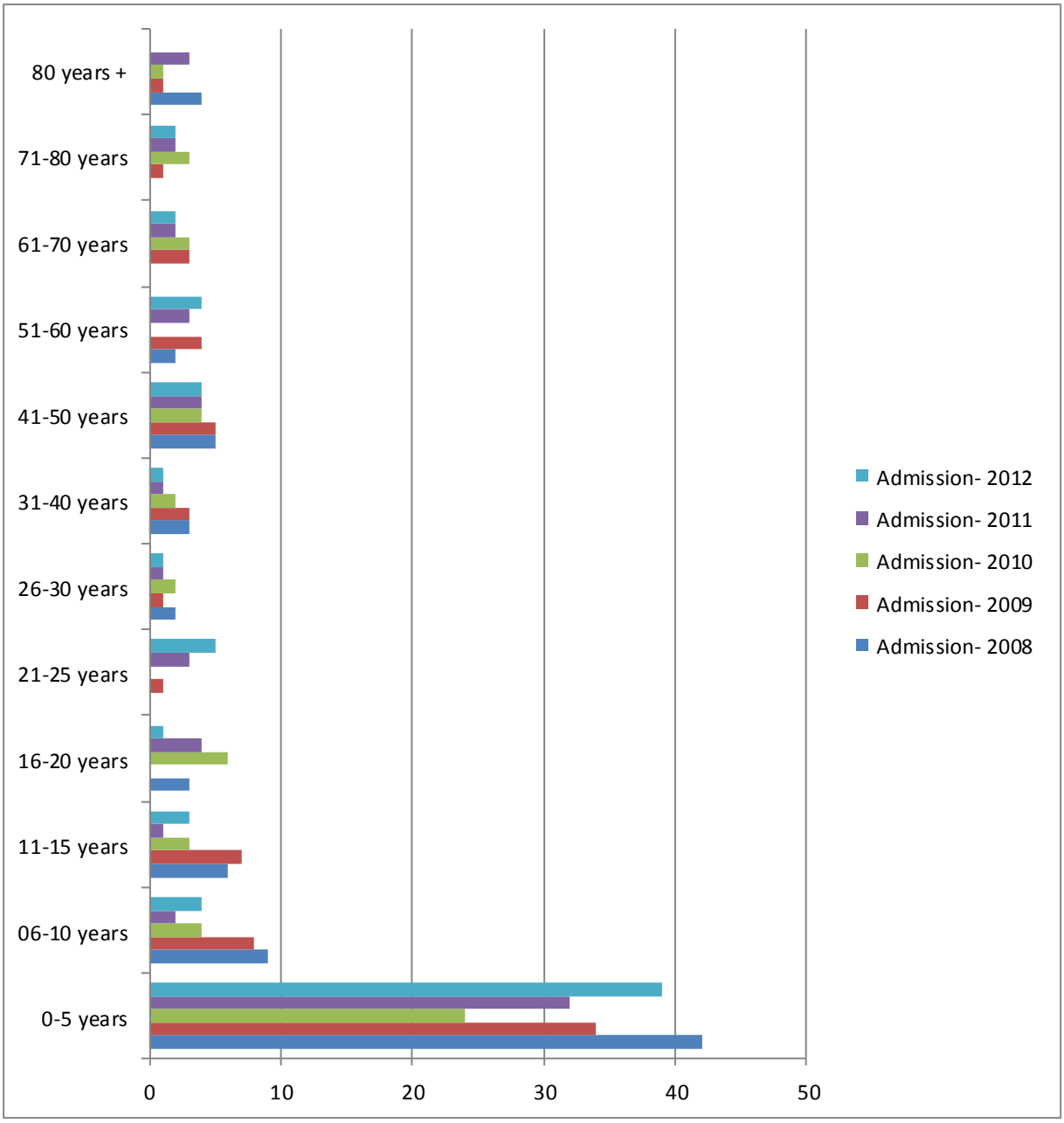


fig. 3.3.5(i) Number of asthma hospital admissions by age for 2008-2012

Figure 3.3.5(i) details the demographic distribution for hospital admissions. In each year more than half of admissions were young children from 0-5 years. This phenomenon was discussed with the clinicians in one of the GP practices who said this would be attributed to the fact that the Primary Care clinicians were less likely to manage a small child with acute asthma symptoms in the community and were more likely to admit that child to hospital than they would with an adult experiencing a similar episode. In addition, families were more likely to take a small asthmatic child to the Accident and Emergency Department (A+E) for admission to hospital whereas adults with asthma would try to manage an asthma attack at home rather than pay up to £145 for a hospital admission. It should be noted that Primary Care services in Guernsey are private. The A+E consultant will also charge a fee for a consultation and admission to hospital. It is only when the patient has been admitted to secondary care that the service will be free.

These figures only provide information on the admissions to hospital and do not reflect the full extent of asthma in Guernsey. Further work will be needed to gain a true picture and this will involve the collection of data from the three private Primary Care practices, which are not linked to a centralised patient health data system at the PEH.

3.4. Temporal distribution of hospital admissions and ambient NO_2 concentrations –lag time trends

From the review of the literature it was noted that asthma patients who were exposed to allergens and concentrations of NO_2 displayed exacerbation of symptoms up to 48 hours after exposure (Rusznak et al, 1996). It therefore follows that it would be useful to examine NO_2 levels on the date of admission and for the period of 48 hours before admission to assess any correlation during the lag time.

As there were only data for real-time ambient concentrations of NO_2 for residential areas in St Peter Port monitored at the Grange monitoring station, the admissions data for that parish has been examined for the years 2010, 2011and 2012. On each date of admission the peak hourly mean concentration for that day has been recorded and then

the peak hourly mean concentration in that period minus 48 hours. The following line graphs show that there was some correlation with this phenomenon. The lower line graph in each case was the peak hourly mean concentration for the date of admission and the higher line graph was the peak hourly mean concentration two days before. It should be noted that on all days the peak hourly mean concentration occurred between 07.30 and 08.30am during the morning work and school traffic jams with two smaller peaks between 3-4pm when school children were picked up from school and later between 5-6pm which were associated with work related traffic flows. There were some limitations with this assessment as it only assessed ambient peak hourly mean concentrations and did not include other impacts from the home or other indoor places. However, the NAQS was based only on ambient concentrations and was therefore useful with the aforementioned caveat.

3.4.1 NO_2 concentrations for the day of admission and 48 hours before admission - 2010

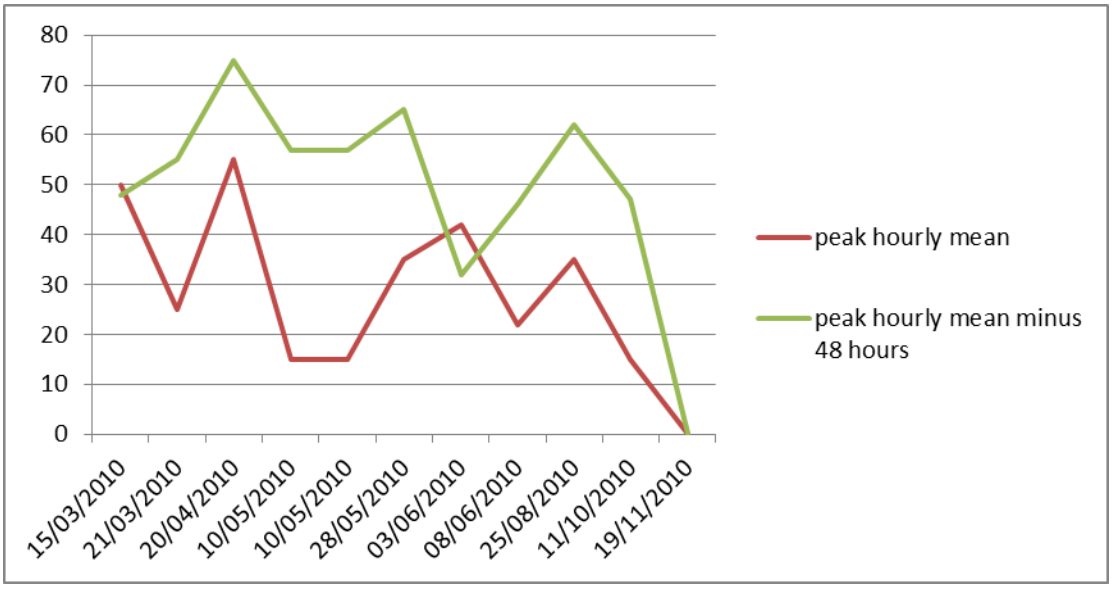


fig. 3.4.1.(i) Admission dates for 2010 and NO_2 concentrations

Figure 3.4.1(i) detailed the variation in NO_2 peak hourly mean on date of patient admission to hospital in 2010 and the peak hourly mean within the preceding 48 hours. This showed that there were generally higher peak NO_2 hourly means in the days

preceding admission rather than on the actual day of admission. This indicated that chronic exposure to NO_2 could have a latent effect rather than acute immediate effect.

3.4.2 NO_2 concentrations for the day of admission and 48 hours before admission 2011

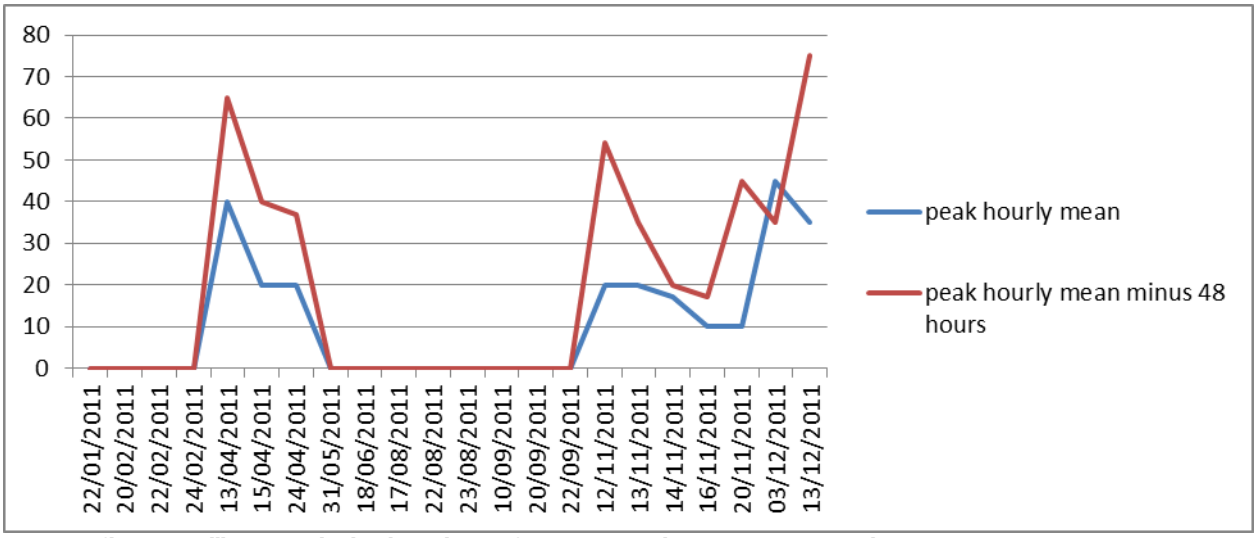


fig. 3.4.2(i) Admission dates for 2011 and NO_2 concentrations

Figure 3.4.2(i) detailed the variation in NO_2 peak hourly mean on date of patient admission to hospital in 2011 and the peak hourly mean within the preceding 48 hours. The flat line area represented the time when data was unavailable due to equipment failure (power cuts/telephone line disconnection etc.). For the records available it can been seen that there was a correlation during 2011, in that there were elevated peak hourly means in the preceding 48 hours before hospital admission.

3.4.3 NO_2 concentrations for the day of admission and 48 hours before admission - 2012

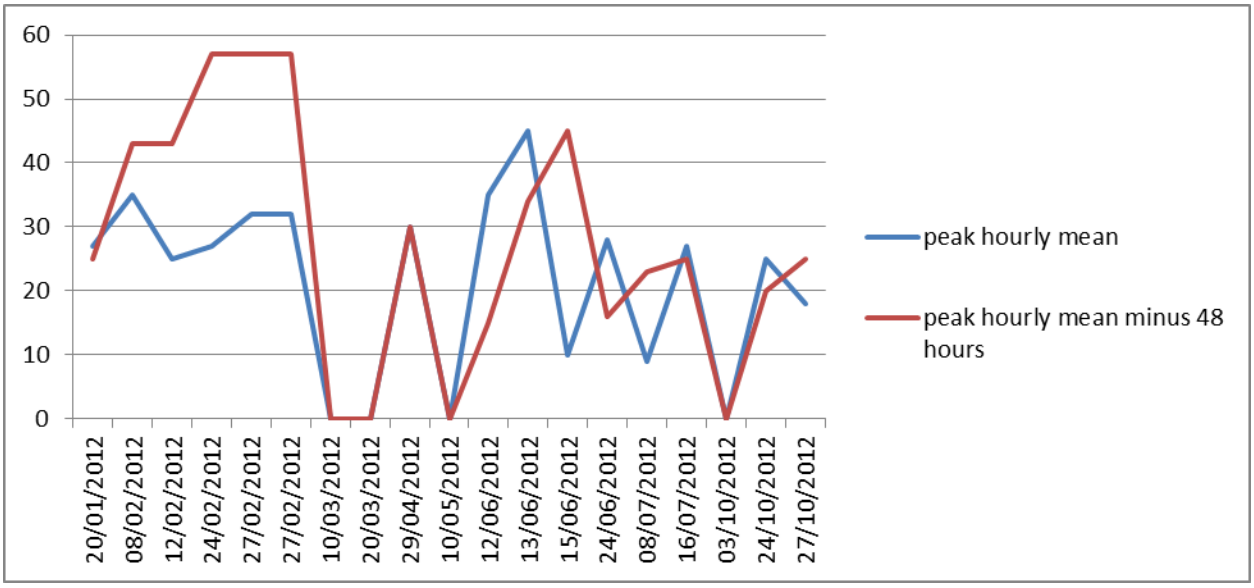


fig. 3.4.3(i) Hospital admission dates 2012 – NO_2 peak hourly mean and minus 48 hours

Figure 3.4.3(i) detailed the variation in NO_2 peak hourly mean on date of patient admissions to hospital for 2012 and the peak hourly mean within the preceding 48 hours**.** In the first quarter of the year the trend generally follows 2010 and 2011 with higher peak hourly means in the preceding 48 hours prior to admission, although the data for later in the year generally replicated the data on the date of admission.

This substantiated the outcomes of the research undertaken by Rusznak et al in 1996, where asthmatic symptoms presented up to 48 hours after exposure to NO_2 and allergens. It was noted that this research involved exposure to allergens and a single high dose of NO_2 up to 400pbb in one hour only, whereas, the data presented in Figures 3.4.1(i), 3.4.2(i) and 3.4.3(i) involved peak hourly means of between 15ppb and 75ppb with exposures over two days before hospital admission.

Therefore, the lag time, or delay, between exposure and presentation of symptoms was an important risk management consideration.

3.5 Conclusion

The aim of this chapter was to identify whether ambient air quality was a good proxy from human health and in particular whether there was spatial and/or temporal correlation or variation in ambient NO_2 levels and impacts on asthmatics.

In this study only those suffering from asthma were considered and not all types of respiratory ill health. However, as asthma was known to be widespread in the population, asthma can be used as an indicator of respiratory health.

A significant problem with this analysis and evaluation was that the numbers of admissions to hospital were very low and needed to be considered with caution. An important consideration was that only very severe cases of asthma were admitted to hospital, with an unknown number of asthma sufferers being managed in the community and not included in this data. This will be another area for research if the true number of asthma sufferers in Guernsey is to be known and this is discussed in the following chapters.

The studies in this chapter focussed on ambient NO_2 concentrations because this was the only NAQS objective in Guernsey to fail the standards set in the UK, which were used for benchmarking purposes.

The studies identified a general temporal correlation in the trend between NO_2 levels and hospital admissions on visual assessment of the trends shown on the various graphs produced. However, statistical calculations revealed little or no correlation.

From the trend graphs, ambient NO_2 concentrations rose, hospital admission numbers increased and conversely, as NO_2 concentrations fell, the number of admissions to hospital fell. It was noted that there has been a general upward trend in the number of admissions to hospital from 2008 to 2012 and that a 48 hour lag time could be an important factor when undertaking an exposure assessment of an individual. There was temporal variation in NO_2 concentrations during the year across all monitoring locations.

The studies revealed that spatial factors were important considerations e.g. the monthly and annual mean concentrations for NO_2 in Fountain Street in St Peter Port frequently exceeded the NAQS standards.

Therefore, it could be concluded that ambient air quality data in Guernsey could be a proxy for respiratory health and should be used as a factor in the risk management of people with asthma.

Chapter 4

Asthma patient study

Overview

This chapter discusses the development of the patient participation questionnaire and selection. There were a number of challenges in completing this work which are described.

The chapter discusses the outcomes of the asthma patient survey and evaluates the correlation between participant responses and NO_2 air quality data for the same study period in Guernsey.

4.1 Introduction

The literature revealed that asthma affects approximately 1 in 10 people (Asthma UK, 2008), which is a significant proportion of the population. In Guernsey, health conditions tend to mirror the UK trends in population health. The population of Guernsey is about 63,000 and so it could be reasonably assumed that about 6,300 people suffer from the symptoms of asthma. During the term of the research programme, the actual number was difficult to determine as the patient health recording systems in Guernsey were split between the Health and Social Services Department, which commissioned secondary care i.e. the hospital and medical specialist services, and three Primary Care GP practices that each had a dedicated patient recording system. Whilst work was ongoing to address this issue, there was no clarity of data between primary and secondary care systems.

A future study will be developed to seek information from all four parties to assess the real impact of asthma in Guernsey and to work together on a compatible data collection system for the island.

The studies undertaken in Chapter 3 indicated that the annual trend of admissions to hospital between 2008 and 2012 was rising year on year.

In order to gain an understanding of the relationship between asthmatic symptoms and NO_2 concentrations, five case studies volunteered to participate in a symptom survey by questionnaire during November 2013. The answers to the questionnaire about the day on which symptoms occurred were then compared with ambient NO_2 concentrations measured by real-time analysers in Guernsey to determine any correlation.

The review of the literature and discussion with relevant colleagues revealed that no other studies had asked the same questions in this context in Guernsey and so this study was novel.

4.2 The survey design

The survey form was designed using the basic principle that it should provide the information required by the study (Harvard University, 2013), in this case the asthma symptoms of participants during the study period. The symptoms selected were wheeze and tightness of the chest and then the possible consequential outcomes i.e. increased use of medication or hospital admission. The participants were also asked what they thought caused worsening of their condition.

The survey was designed to be anonymous, with only reference to postcode of residence and postcode of workplace being spatial locators.

In order to support the comparison with NO_2 levels, the days of the month that symptoms occurred was also needed. The survey format was a consideration to ensure the required outcomes. It was decided that a self-completion daily diary should be completed by the study participants. This method has been well used in health surveys and provides real-time information that participants can record at the time and which they may not be able to recall at a later stage if an interview style of survey was conducted (Verbrugge, 1980).

It is known from the literature that asthma is triggered by an allergen (Asthma UK, 2013) so some basic information about the participant's triggers was required.

The participants were asked to state their age and basic information about whether they smoked, or lived with smokers, had pets living in their house and which allergies they suffered from. These confounding factors were personal to the individual participants involved and would have an impact on their symptoms during the survey study period.

The survey form was designed to ask some very simple questions about the days on which the participant felt wheezy and when they had increased their usual medication to improve their health status. In addition, the participants were asked to state any days on which they had been admitted to hospital (ECRHS, 1996).

The answer to each question was recorded by the participant by simply circling the date on a grid numbered from 1 to 31, for the days of the month (only numbers 1 to 30 were needed for November).

The survey form and participants information letter etc. can be found in Appendix 4.

4.3 Ethics Approval

The original ethics approval was granted by the Ethics Committee at Abertay University during the early stages of the research programme. However, having moved to Guernsey, further approval was sought from and granted by the Ethics Committee of the Health and Social Services Department in Guernsey.

4.4 Outcomes of the survey

A survey was initially carried out in 2007, prior to the move to Guernsey and was therefore not suitable for comparison with NO_2 concentrations data in Guernsey.

The study was again undertaken in 2012 in Guernsey, although the participants failed to complete the questionnaires.

In 2013, following consultation with the Respiratory Nurse Consultant and further ethics approval, the survey was undertaken again.

Five participants volunteered to undertake the survey during November 2013.

Of the participants, three (60%) lived in St Peter Port and two (40%) lived in St Pierre du Bois, a rural parish. Three (60%) of the participants were female and two (40%) were male. The age range of the participants was 16-29 years so were ‘children of the incidence era’.

All participants lived in, or travelled to, St Peter Port each day. None of the participants were smokers and two of the participants had pet dogs living in their houses. It was

noted that of the two dog owners, one reported being allergic to cats and both reported allergic reactions to grass/hay fever and dusts.

The other three non-pet owners reported allergies towards pollens, animals, house dust mites, bird feathers and dust.

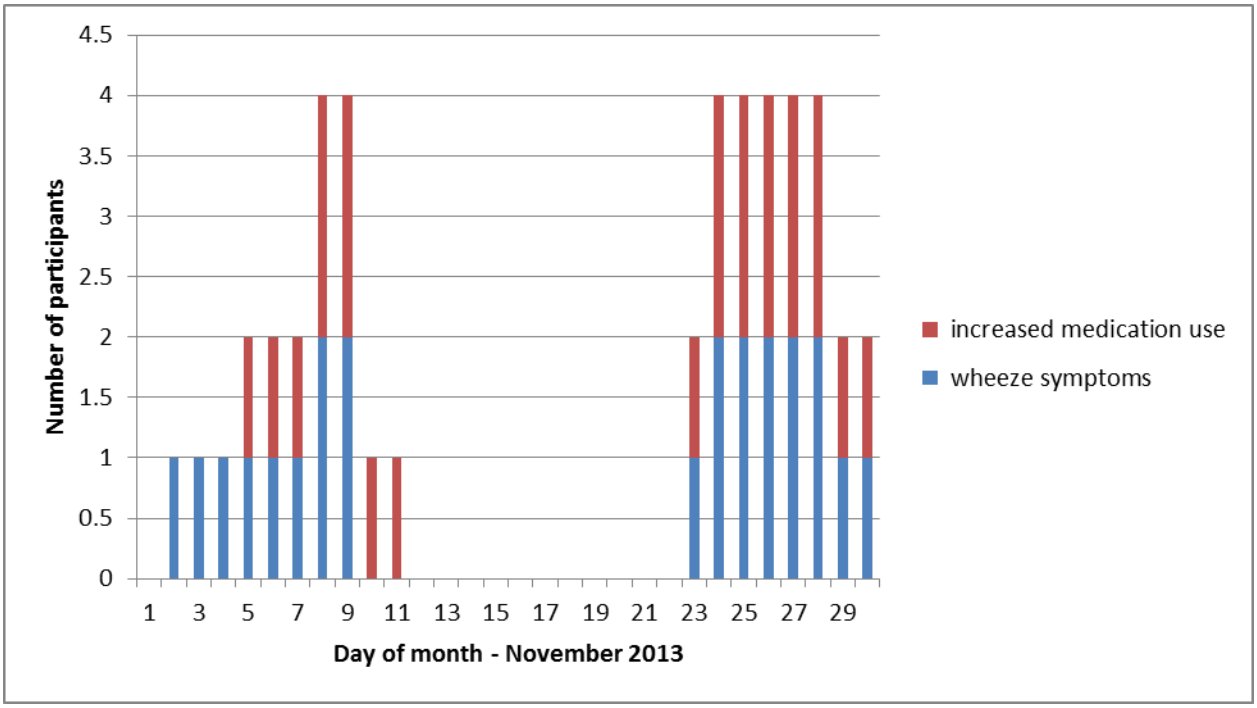


Fig. 4.4(i) Days of asthmatic episodes and increased medication use – November 2013

In response to question 1, 4 out of 5 (80%) of the participants had experienced episodes of wheeze or tightness of the chest during the study period and the same participants had experienced episodes when they had been required to increase their usual medication.

None of the participants had been admitted to hospital during the study period.

In response to question 4 about their perception of what makes their asthma symptoms worse, only 3 participants responded. Each of the respondents were unsure about what made their asthma symptoms worse, but the three who responded said they did agree that the cold (assumed lower temperature) colds and flu or chest infections contributed to worsening ill health. None mentioned air pollution as a contributing factor.

Anecdotally, one respondent stated that that they had lived in London for three years and their asthma had been much more difficult to manage and had resulted in repeated hospital admissions, which they thought was due to the congested traffic. Since living in Guernsey for the last two years their general asthma symptoms had been much improved and easier to manage.

4.5 Asthmatic episodes and NO_2 Concentrations - November 2013

Figure 4.4(i) detailed the days of November when the survey participants experienced wheeze or tightness of the chest and the days on which they increased their use of medication. The daily mean and the peak concentrations of NO_2, were assessed for the days when 2 participants experienced symptoms to establish whether there was a temporal correlation.

Due to failure of the electronic connection from the monitoring station to the OEHPR, only data for 24-28th November could be analysed.

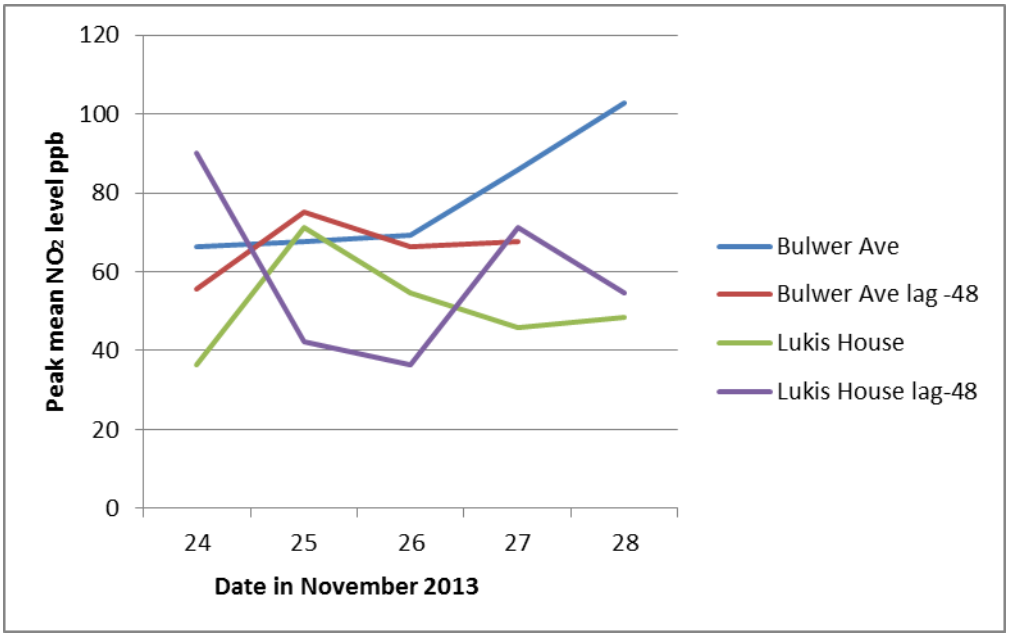


fig. 4.5(i) NO_2 concentrations in ppb on days when participants experienced symptoms and increased medication use.

Figure 4.5(i) indicated that the daily mean and peak NO_2 concentrations during the study period were significantly different. This temporal variation in NO_2 levels occurring though the day with significant peak levels would have an impact on the daily exposures of asthmatics and coincided with the days they were experiencing episodes of ill health and increased use of medication. The peak concentration for the 48 hour period before showed the concentration during the lag time.

4.6 Conclusion

The qualitative data gathered during this survey provided an insight into the complexity of asthma and how that can confound exposure assessment and effective risk management of individuals.

This small study of five people revealed that they were all unsure about what made their asthma worse although exacerbation of symptoms was attributed to colds and infections which would increase mucous production. None of the participants considered air pollution as an impact on their health.

This study was very small and will be worthy of further research in the future using a larger cohort.

Chapter 5

Indoor NO_2 levels and exposure assessment

Overview

During November 2011 and March 2012 the indoor NO_2 concentrations in homes and workplaces were monitored using diffusion tubes. The aim of this study was to assess whether concentrations of NO_2 in indoor environments were comparable with ambient NO_2 concentrations.

In addition, this study aimed to assess whether micro environments exist for NO_2 and whether this needs to be considered in exposure assessment.

Indoor micro environments may be affected by indoor air flows, so this study modelled indoor air flows to assess the spatial distribution of NO_2.

The study was further validated by real-time monitoring of NO_2 at the study locations as described in Chapter 2.

The 24 hour exposure assessment of three case studies was undertaken using mean NO_2 concentration data.

5.1 Introduction

The aims of this Chapter were to examine and explore aim numbers 2, 3 and 5 as detailed in Chapter 1.

- To evaluate multiple exposures and how multiple exposure assessment can be used in conjunction with the ambient air quality measurements to identify spatial and temporal variations.

- To identify whether micro-environments for NO_2 exist in buildings and whether building design and layout play a role in exposure assessment.

- To assess whether exposure to NO_2 is a necessary characteristic in risk assessment of pre-existing asthmatics in indoor workplaces.

5.2 Indoor NO_2 concentrations observed in workplaces and homes- diffusion tube study

This section provides an overview of the concentrations of NO_2 that were observed in three houses and four workplace locations in order to assess whether there was any correlation with ambient air concentrations. These data provided the spatial and temporal distribution of NO_2 in the study locations described in Chapter 2, method 2.

5.2.1 House 1

Figure 5.2.1(i) detailed the NO_2 concentrations observed as monthly mean values sampled using passive diffusion tubes. It can be seen that the NO_2 levels in the property were similar with spatial and temporal variation of 6.1ppb to 10 ppb, whereas outside the ambient temporal variation was from 6.1ppb to 14.2ppb. This property was close to the Grange, one of the main feeder routes into St Peter Port.

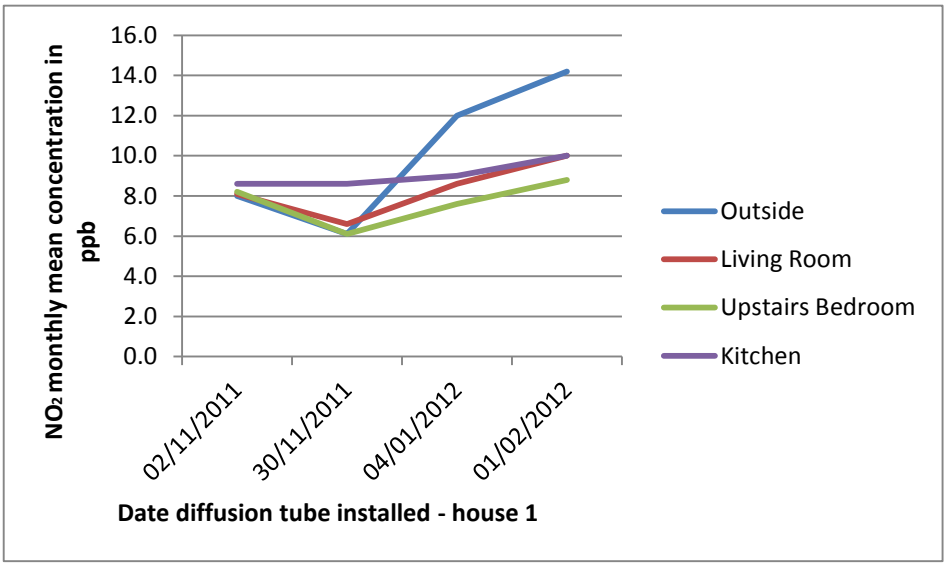


fig. 5.2.1(i) NO_2 monthly mean concentrations (ppb) in house number 1

5.2.2 House 2

Figure 5.2.2(i) detailed the observed monthly mean concentrations for NO_2 measured in ppb by diffusion tube. This graph showed that the indoor concentrations followed a consistent trend with a variation between 2.2ppb and 9.3ppb. During the sampling period there was a slight increase from November to March, with a sharp dip in ambient concentrations in December. This was likely to be a correlation with reduced work and school traffic flows over the Christmas holidays. Interestingly, the NO_2 levels in the garage where the central heating boiler was located showed concentrations above ambient levels during December and January, when the weather is colder and the boiler was potentially running at a higher capacity.

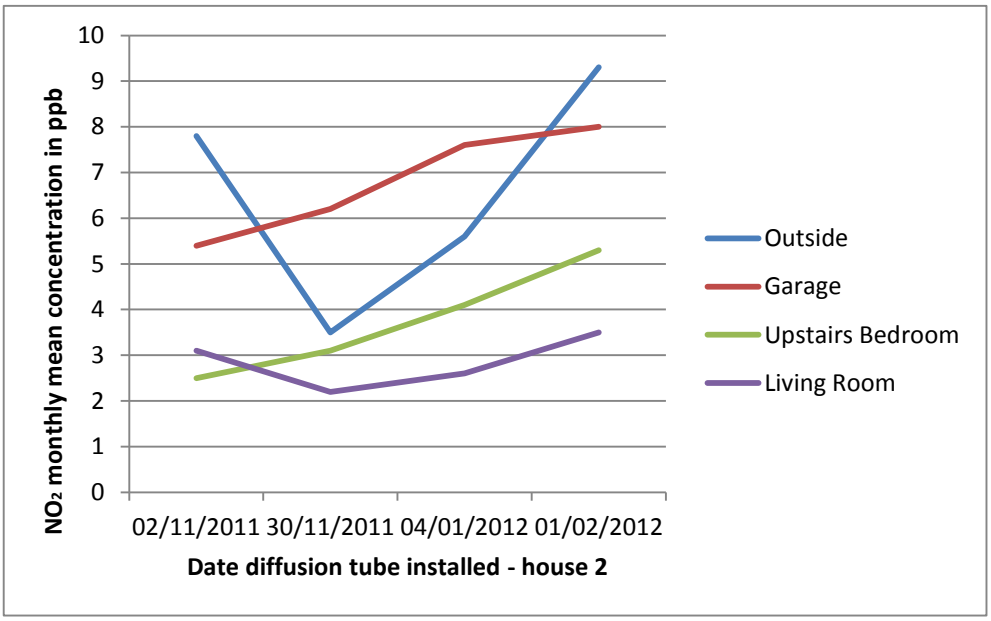


fig. 5.2.2(i) NO_2 monthly mean concentrations (ppb) in house number 2

5.2.3 House 3

Figure 5.2.3(i) detailed the observed NO_2 monthly mean concentrations measured in ppb by diffusion tube for house 3. This graph indicated that the NO_2 concentrations in this property were generally lower than in house numbers 1 and 2, which is due to its location in Castel, one of the rural parishes. Here the concentrations vary from 6.1ppb to14.2ppb. This property had an open coal fire in the living room and a gas fired central heating boiler in the kitchen. This indicated that open combustion processes contribute to higher concentrations of NO_2 in indoor spaces, particularly in February during colder weather. As with house numbers 1 and 2 there was a dip in ambient NO_2 levels during December and also in the living room and bedroom although the levels in the kitchen where the gas fired central heating boiler was located, maintained a more consistent concentration throughout the study period with a temporal variation of 1.4ppb.

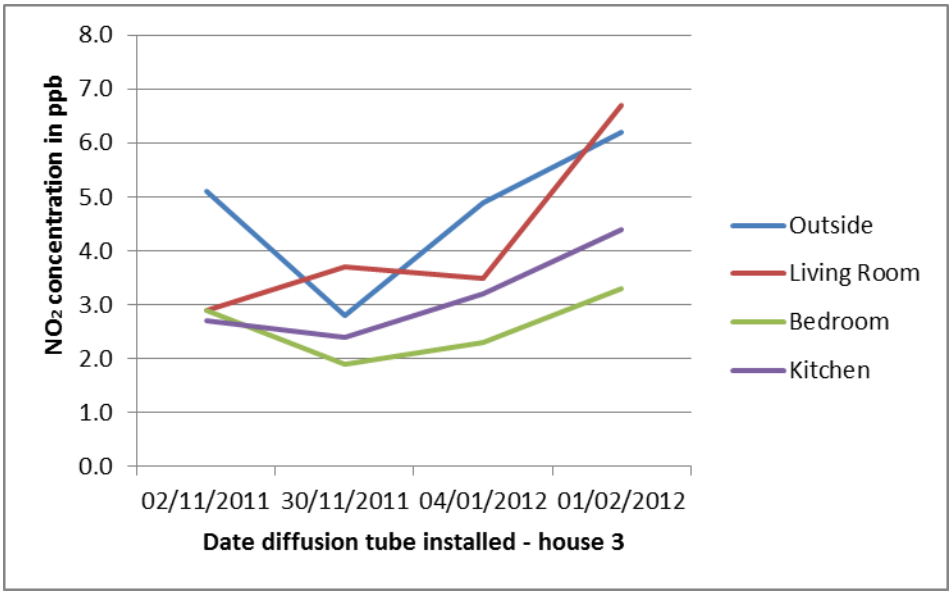


fig. 5.2.3(i) NO_2 monthly mean concentrations (ppb) in house number 3

5.2.4 Workplaces

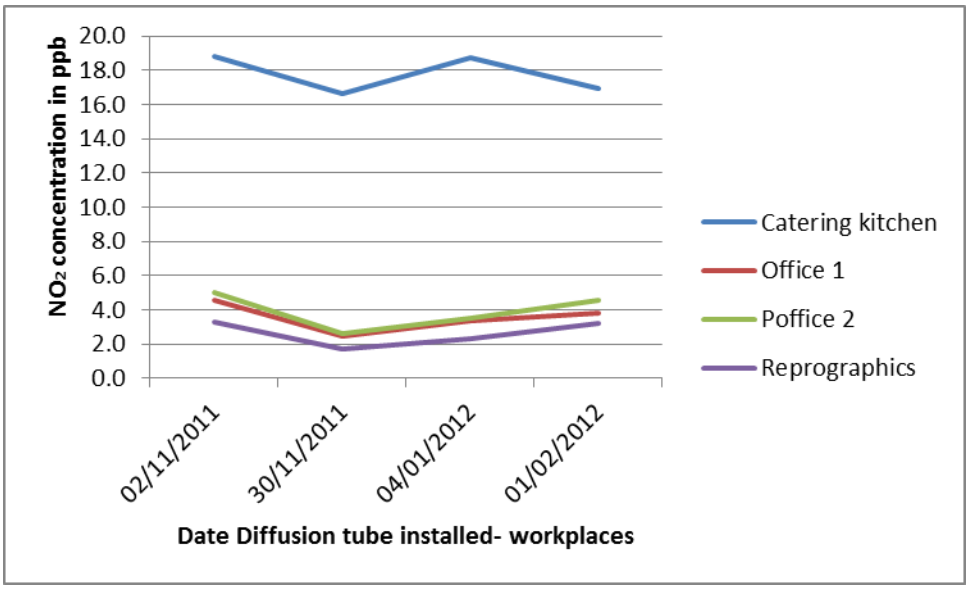


fig. 5.2.4(i) NO_2 monthly mean concentrations in workplaces at the Princess Elizabeth Hospital

Figure 5.2.4(i) detailed the NO_2 monthly mean concentrations measured in ppb using diffusion tubes for four different workplace locations at the Princess Elizabeth Hospital. The two different offices and the print room followed a consistent trend with very low concentrations with variability of 1.7ppb to 5ppb. The monthly mean concentrations for the catering kitchen were higher, with variability between 16.6ppb and 18.8ppb.

Diffusion tubes for ambient measurements were not included in this part of the study as there was already an ambient diffusion tube monitoring site in the grounds of the PEH. During this sampling period ambient NO_2 levels were 6.7ppb (30.11.2011), 2.2ppb (04.01.2012), 5.1ppb (01.02.2012) and 7ppb (29.02.2012). This trend shows that the ambient levels followed a consistent concentration to the two offices and the print room, being only slightly higher than the indoor levels. However, in the catering kitchen, the NO_2 concentrations were significantly higher than the ambient levels in the area of the Princess Elizabeth Hospital.

This information indicated that indoor combustion processes in an indoor workplace contributed significantly to NO_2 levels and these were significantly higher than ambient NO_2 levels. Therefore, indoor combustion processes will be an important consideration when developing a risk management framework for asthmatics.

5.3 Indoor NO_2 concentrations observed in workplaces and homes – Co-location real-time gas analysis study

Whilst diffusion tube surveys have their merits in providing general data about the environment being studied, the data is an average of the concentrations in that environment during the exposure period and cannot provide data on the peak concentrations that may have occurred and therefore the peak exposures individuals may have experienced in those environments. During the day and night there will be variations in concentrations, especially in connection with the use of combustion appliances such as open fires, gas and oil central heating boilers, gas cookers etc. There will also be seasonal variation for these parameters too. To develop a risk management framework, it will be necessary to consider the 'worst case scenario' to ensure that asthmatics have the best protection in the environments they work in.

In order to assess the actual levels of NO_2 in the study locations, a Gray Wolf gas detector was used. The main concern with this methodology was the amount of time that the study would take because the equipment was very expensive and needed constant supervision, and was also needed by the staff of the OEHPR during their routine duties. In addition, regular access to houses 1 and 2 proved difficult, so only house number 3 was sampled.

In each of the study locations, NO_2 was measured for 2 minutes every 10 minutes for a period of 1 hour. The six samples were then averaged to provide the hourly average and to allow comparison with the diffusion tube data. The measurements were taken during February 2012.

From the review of the literature and previous experience, the timing of the sampling period varied to allow for the 'worst case scenario' e.g. in the house this was during the evening when the open fire was operating and the gas central heating boiler was working, then during the morning when the catering kitchen was being used for cooking. The offices and print room were sampled during the day over the lunch time periods so as to reduce disruption to staff working.

Site	NO_2 ppb at 0- 2 minutes	NO_2 ppb at 10- 12 minutes	NO_2 ppb at 20- 22 minutes	NO_2 ppb at 30-32 minutes	NO_2 ppb at 40-42 minutes	NO_2 ppb at 50-52 minutes	Average ppb
House 3 Living room	17	15	18	17	12	17	17
Office 1	10	9	9	11	10	9	9.6
Office 2	8	8	6	8	6	6	7
Print room	12	12	14	13	11	13	12.5
Catering kitchen	130	127	129	129	128	127	128

table 5.3(i) real time NO_2 concentrations at study locations measured during February 2012

Table 5.3(i) provides the details of the observed NO_2 concentrations measured in ppb using the Gray Wolf gas detector, at the study locations. The average concentrations gave an indication of the typical exposures to NO_2 in the home and workplace.

The data identified that the average measured concentration of NO_2 in house 3 during the evening was 17ppb, which was higher than the monthly mean measured during February 2012 by diffusion tube, which was 6.7ppb in the same living room. The ambient NO_2 monthly mean for the same period at house 3 was 6.2ppb. These data identified that the NO_2 concentrations measured in real time were higher during the evening period when the open fire was in operation, than the monthly mean

concentration measured by diffusion tube. This was probably due to residents working through the day and then lighting the fire in the evening when they were at home.

For the 2 offices and the print room the average measured level of NO_2 during the real-time sampling period was slightly higher than the monthly mean concentration measured by diffusion tube during February 2012.

The data for Office 1 showed the measured average concentration of NO_2 was 9.6ppb during the hour of sampling compared with the diffusion tube survey of 4.6ppb measured as the monthly mean in February 2012. The ambient monthly mean for the same sampling period was 7ppb.

The data for Office 2 shows the measured average concentration of NO_2 was 7ppb during the hour of sampling compared with the diffusion tube survey of 3.8ppb measured as the monthly mean for February 2012. The ambient monthly mean for the same sampling period was 7ppb.

The data for the print room showed higher concentrations of NO_2 with an average concentration of 12.5ppb during the sampling period compared with the monthly mean concentration for February 2012 of 3.2ppb for the print room. It was noted that the service was operating on a part time basis and the room was locked for part of the day reducing ventilation. The ambient monthly mean for the same sampling period was 7ppb.

The catering kitchen data showed the most significant variation in NO_2 concentration. The average measured level of NO_2 during the hour of sampling was128ppb during the cooking process which was significantly higher than the 16.9ppb monthly mean measured by diffusion tube during February 2012 The ambient monthly mean for the same sampling period was 7ppb.

This variation indicated that during cooking the NO_2 levels were much higher than anticipated when considering the monthly mean. It was noted that cooking operations only take place on five or six days of the week and most of the cooking operation takes three to four hours on each day.

This study, whilst limited in the time period and frequency of sampling, indicated that there could be significant exposures for asthmatics if working in catering establishments where gas cookers were used. The study identified that there were variations in concentrations of NO_2 when measured in real time and when NO_2 was measured as a monthly mean by diffusion tube. Therefore, real-time analysis of NO_2 concentrations will be an important consideration when combustion processes are present in indoor environments and when developing a risk management framework for pre-existing asthmatics at work.

5.4 Building design and air flow

During the review of the literature it was well established that indoor air flows play an important role in ventilation of indoor spaces and the development or indeed, the prevention of indoor micro environments (Council Directive 89/106, 1989; Delfino, 2008; COMEAP, 2004; HSE, 2003). Good building design and layout would be crucial characteristics for the even dispersion and removal of indoor pollutants, including NO_2.

In order to test this hypothesis, an air flow model for a room in each of the house study locations and the two offices was undertaken using a simple airflow modelling tool called ‘Flowsquare’ (refer to section 2.6.1, method 3).

The room layout was entered into the model, identifying the locations of door and windows to give a representation of the air flow for that room. The model shows the air flow by direction using arrows, which vary in size depending on the strength of the air flow. The model also indicated the airflow by colour, red being the higher velocity through to orange, yellow and green to blue where there was little or no air movement.

5.4.1 Air flow model for house number 1

House number 1 was a small terraced property. To assess indoor airflows in house number 1, the ground floor was modelled which included the living room, kitchen and access to the open staircase. The living room had a window and was divided from the

kitchen by a party wall with a door. The main entrance door opened into the kitchen area, and there was a window in the kitchen. The stairwell to the first floor opened into the kitchen area. This area was selected because the monitoring data indicated this area displayed a higher concentration of NO_2 than other areas of the house.

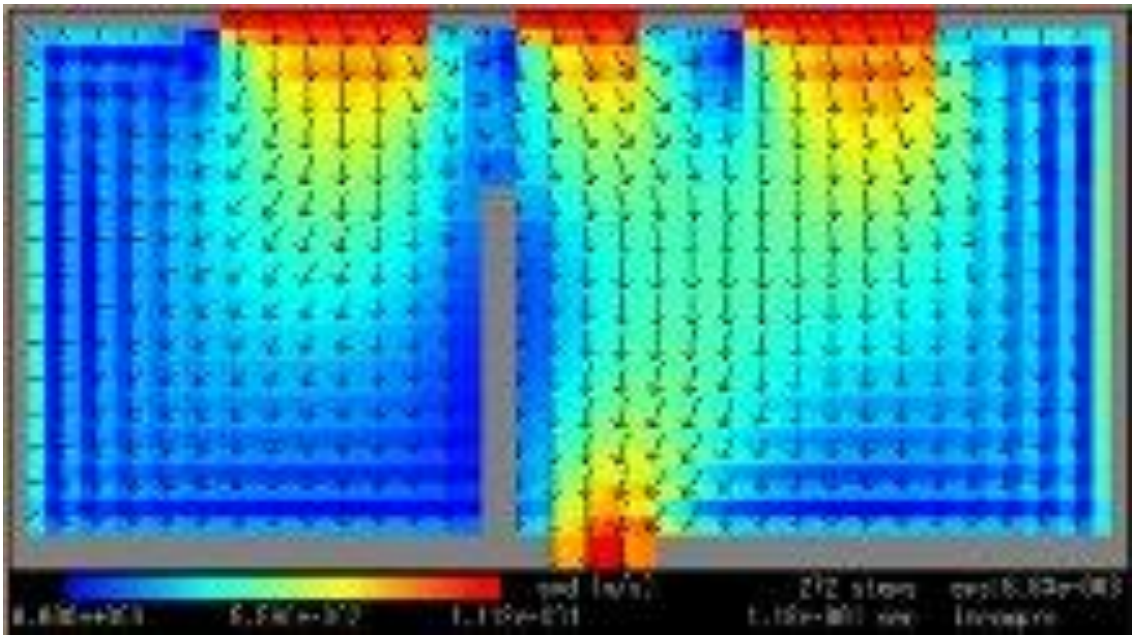


fig. 5.4.1(i) Air flow model of house number 1 –ground floor

Figure 5.4.1(i) detailed the air flow in house number 1. The air circulation was greatest in the areas around the windows and door openings and adjacent to the open stairwell where model was coloured red and orange. The corners of the rooms diagonally opposite the windows and door opening were shown in blue and therefore little or no air movement. This indicated that there was potential for indoor micro environments to develop if there was insufficient ventilation.

5.4.2 Air flow model for house number 2

House number 2 was a large property and the garage/utility room was selected for the air flow modelling process because the monitoring data for NO_2 in house number 2 indicated that this area displayed the highest concentration in the property due to the

location of the oil fired central heating boiler. The utility room was accessed from the main kitchen and was separated from the garage by a party wall with a door. The party door between the garage and utility room was generally kept closed by the occupants.

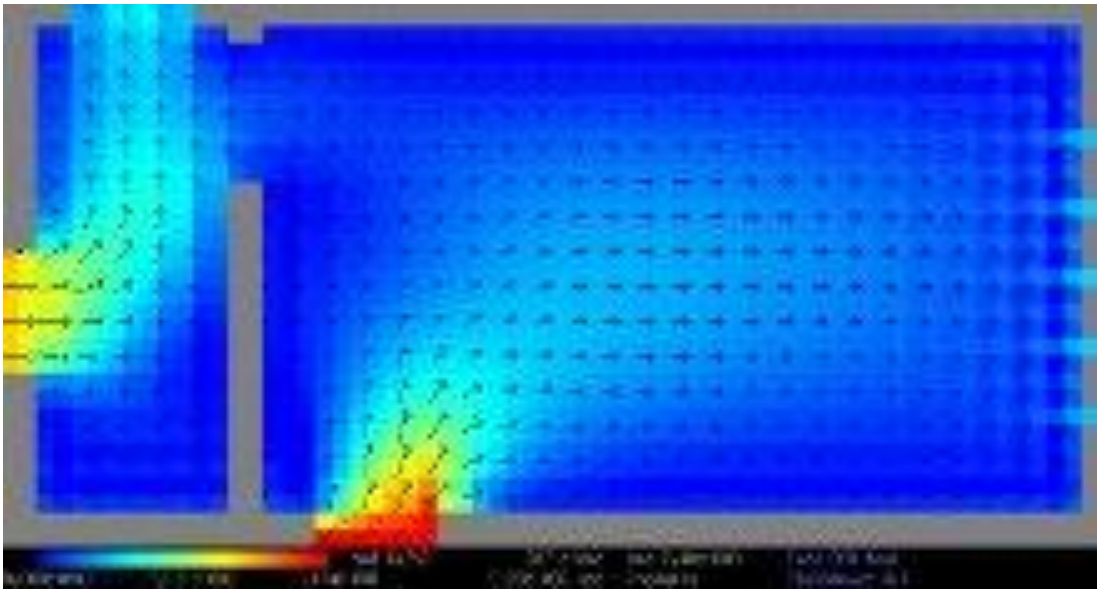


fig. 5.4.2(i) Air flow model for house number 2 – garage/utility room

The model detailed in figure 5.4.2(i) indicated that the air flow into the utility room was from the kitchen area and towards the door opening into the garage. This air flow being yellow and green in colour is of moderate strength, leading to poor or no air movement in the corners shown in blue. In the garage the small window opening facilitated increased air flow in the vicinity of the window, but little or no air flow in the corners of the garage area. This clearly indicated that a micro environment developed in the garage area.

5.4.3 Air flow model for house number 3

House number 3 was a bungalow with an open fire in the living room and a gas-fired central heating boiler in the kitchen. For this study, the kitchen and living room were selected as the data for these areas displayed the highest concentrations of NO_2 in that

property. The kitchen had a window opening and the rear entrance door to the property opened into the kitchen. The kitchen connected to the living area through a door opening in the party wall. The living room had two large windows and an internal door leading to the hallway.

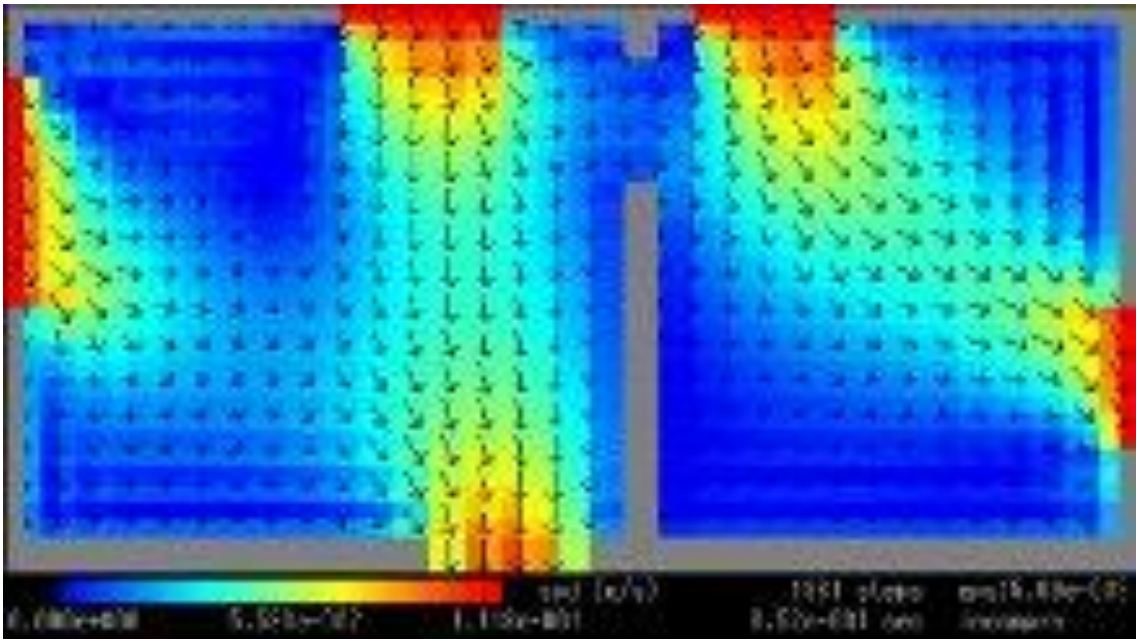


fig. 5.4.3(i) Air flow model for house number 3 –kitchen/living room

The air flow model detailed in figure 5.4.3(i) showed a diagonal air flow in the kitchen area, to the right, between the window and the rear entrance door highlighted in red and orange adjacent to both apertures with reducing velocity in the middle of the room. The air movement in the two opposite corners was much reduced being shown in blue with little or no air movement.

The living room on the left of figure 5.4.3(i) showed two main air flows, the main being between the large window and the doorway to the hall, the second flow was between the main window across the centre of the room towards the hall doorway.

5.4.4 Air flow model in Office 1

Office number 1 was located in the PEH building. The office included two openable windows, one fixed window and an entrance door onto the main corridor.

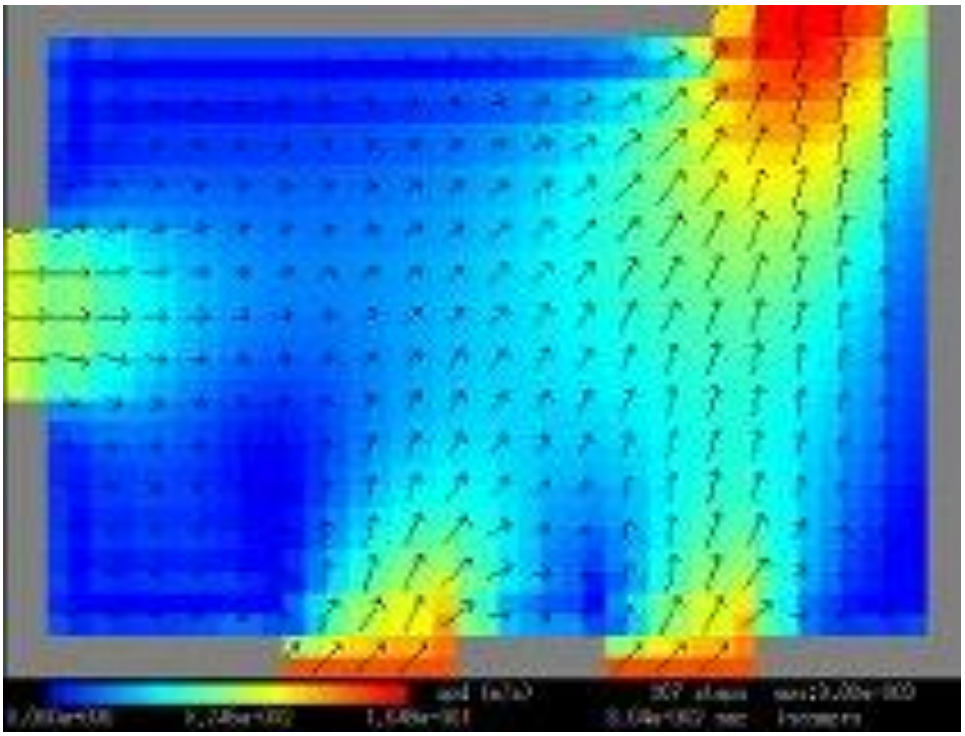


fig. 5.4.4(i) Air flow model for Office 1

The main air flows detailed in figure 5.4.4(i) were between the openable windows towards the door to the corridor. The model indicated that the air flow velocity was greatest near the window openings and door entrance. The model highlights that the fixed window had an impact on indoor air flows even though it was fixed due to convection created by heat from the sun.

5.4.5 Air flow model for Office 2

Office number 2 was located in the PEH. The office had one door way which was entered through another office, one openable window and another door that connected to the main corridor but was usually kept locked.

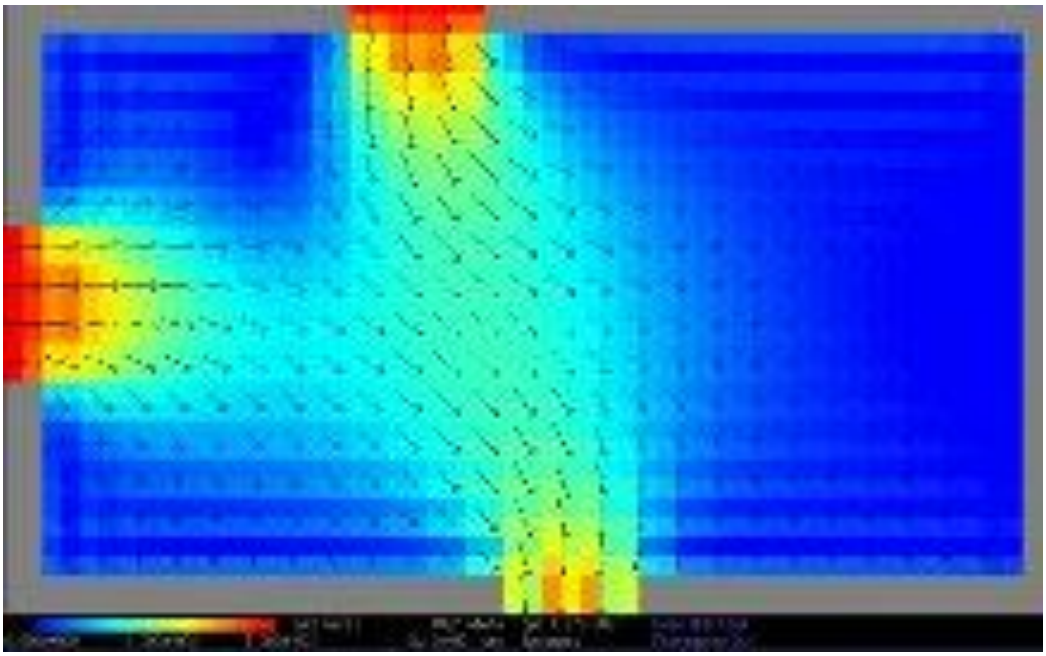


fig. 5.4.5(i) Air flow model for Office 2

The air flow model in figure 5.4.5(i) highlights that the greatest air velocity was located near the openable apertures, although the locked door contribute to the air flow in the room to a lesser degree in that air flowed from the openable door and window towards the locked door and out to the corridor. This model shows that about one third of the room has little or no air flow and therefore would be prone to the creation of an indoor micro environment. It was noted that the workstation of the occupant was located in the corner of the room furthest away from the openable door and window.

The five air flow models provided information to validate that building layout and design can have a direct impact on air flow and ventilation and therefore it can be assumed, the respiratory health and wellbeing of occupants. The rooms in the residential study locations and the office locations varied in size and layout and there was a significant variation in air flow between each building layout. Increased air flow occurred between openable doors and windows, with decreased or no air flow in the corners of rooms away from openable apertures.

5.5 Spatial distribution of NO_2 in house number 3

This study was undertaken to assess the spatial distribution of NO_2 in house number 3 following the air flow modelling exercise to assess whether there was any correlation and to establish whether indoor micro environments for NO_2 had developed. House number 3 was selected for the study because it had low monthly mean concentrations of NO_2 measured by diffusion tube and greater variation in concentrations when measured by real-time analyser.

This study required the deployment of the Gray Wolf gas detector to measure the NO_2 concentrations in ppb in real time. One minute samples were recorded at 300mm space intervals horizontally and vertically at a height of 300 mm, 600mm, 900mm, 1200mm, 1500mm. The survey took place over five consecutive days in February 2012 (refer to section 2.3.5.2, method 3).

During each sampling period the doors and windows were closed and the open fire was operating and burning coal and wooden logs. Various stools and step ladders were deployed to ensure a consistent height as the gas detector was moved around the room.

The following tables detailed the outcomes of this survey. In order to provide a pictorial representation of the NO_2 concentrations measured, the excel spreadsheet has been coloured using the same colour scheme as ‘Flowsquare’ for easy comparison as follows:-

5.5.1 NO_2 concentrations 300mm from the floor

8	8	8	8	6	10	11	13	9	8	5	5	5	5	3
9	9	8	8	8	10	11	11	9	7	6	6	5	4	3
9	8	9	8	8	9	10	12	10	7	7	5	5	4	4
7	7	7	7	7	8	9	10	9	6	3	3	2	3	3
5	5	5	5	4	4	6	7	8	6	3	2	3	3	3
3	3	3	4	4	5	3	3	4	3	3	2	2	2	3
1	3	4	5	3	4	3	4	6	4	3	3	3	2	3
2	3	5	4	5	4	4	3	3	3	3	3	2	4	4
2	3	3	4	3	3	4	4	3	3	3	3	2	3	4
4	6	4	4	3	3	4	3	3	3	3	3	2	3	3
5	6	5	4	3	4	4	4	3	3	3	3	2	4	4

fig. 5.5.1(i) NO_2 concentrations at 300mm from the floor

Discussion

Figure 5.5.1(i) indicated the NO_2 concentrations measured in ppb across the floor of the room at a height of 300mm. The data showed a variation in NO_2 concentrations in the room with higher concentrations being recorded near the open fire and lower concentration being recorded near the door and window apertures.

The distribution of NO_2 indicated that slightly elevated concentrations were measured in the corner to the top left of the room and much lower levels to the bottom right of the room which was adjacent to the doorway to the hall.

The spatial variation in NO_2 concentrations was 1-13 pbb and the data showed that the NO_2 concentrations in the room at 300mm vertically was, by visual observation, correlated to the air flow on the left hand side of the room, although showed less correlation in the right hand side.

5.5.2 NO_2 concentrations 600mm from the floor

9	9	8	8	8	10	12	13	11	7	6	6	5	4	3
9	8	9	8	8	9	11	12	10	7	7	5	5	4	4
7	7	7	7	8	8	10	11	10	7	5	4	4	4	3
7	7	7	7	7	8	9	10	9	6	3	3	2	3	3
5	5	5	5	4	4	6	7	8	6	3	2	3	3	3
3	3	3	4	4	5	3	3	4	3	3	2	2	2	3
1	3	4	5	3	4	3	4	6	4	3	3	3	2	3
2	3	5	4	5	4	4	3	3	3	3	3	2	4	4
2	3	3	4	3	3	4	4	3	3	3	3	2	3	4
4	6	4	4	3	3	4	3	3	3	3	3	2	3	3
5	6	5	4	3	4	4	4	3	3	3	3	2	4	4

fig. 5.5.2(i) NO_2 concentrations at 600mm from the floor

Discussion

Figure 5.5.2(i) indicated the NO_2 concentrations measured in ppb across the floor of the room at a height of 600mm. The table showed a variation in NO_2 concentrations in the room with higher concentrations being recorded near the open fire and lower concentrations being recorded near the door and window apertures.

Although the NO_2 variation was between 1-13ppb, the area in front of the open fire experienced a greater frequency of the higher concentrations than at 300mm from the floor. The same distribution in the top left hand corner was demonstrated indicating that a micro environment of slightly elevated levels of NO_2 from 8-9ppb existed in that corner.

The distribution of varying concentrations of NO_2 across the room was roughly the same as at 300mm vertical.

5.5.3 NO_2 concentrations 900mm from the floor

9	8	9	8	8	10	11	12	10	8	7	5	5	3	3
7	9	7	7	8	9	10	11	10	8	5	4	3	3	3
7	7	7	7	7	9	10	10	9	7	3	3	2	3	3
5	5	5	5	4	6	6	7	8	6	3	2	3	3	3
3	3	3	4	4	6	5	5	5	3	3	2	2	2	3
1	3	4	5	3	5	6	4	6	4	3	3	3	2	3
2	3	5	4	5	4	4	3	3	3	3	3	2	4	4
2	3	3	4	3	3	4	4	3	3	3	3	2	3	4
4	6	4	4	3	3	4	3	3	3	3	3	2	3	3
5	6	5	4	3	4	4	4	3	3	3	3	2	4	3
6	6	6	5	5	3	4	3	3	3	2	2	2	3	3

fig. 5.5.3(i) NO_2 concentrations at 900mm from the floor

Discussion

Figure 5.5.3(i) indicated the NO_2 concentrations measured in ppb across the room at a height of 900mm. The table showed a spatial variation in NO_2 concentrations in the room with higher concentrations of 10-12ppb being recorded near the open fire and lower concentrations of 1-4ppb being recorded near the door and window apertures.

The height of 900mm was roughly the same height that the occupants of the house were breathing at, when sat down in the room e.g. watching television.

The spatial variation in NO_2 concentrations was 1-12ppb and the area in front of the open fire indicated that elevated concentrations were experienced there, as compared with the areas of the room in close proximity to window and door apertures.

5.5.4 NO_2 concentrations 1200mm from the floor

8	8	9	9	10	12	11	11	9	8	5	5	5	4	3
9	9	9	9	8	10	11	11	9	7	6	6	5	4	3
9	9	9	8	8	9	10	10	10	7	7	5	5	4	4
7	7	8	7	8	8	9	9	9	7	5	4	4	4	3
5	5	5	5	4	4	6	7	8	6	3	2	3	3	3
3	3	3	4	4	5	3	3	4	3	3	2	2	2	3
1	3	4	5	3	4	3	4	6	4	3	3	3	2	3
2	3	5	4	5	4	3	3	3	3	3	3	2	3	3
2	3	3	4	3	3	4	3	3	3	3	3	2	3	3
4	5	4	4	3	3	4	3	3	3	3	3	2	3	3
5	5	5	4	3	4	4	4	3	3	3	3	2	4	4

fig. 5.5.4(i) NO_2 concentrations at 1200mm from the floor

Discussion

Figure 5.5.4.(i) indicated the NO_2 concentrations measured in ppb across the floor of the room at a height of 1200mm. The table showed a variation in NO_2 concentrations in the room with higher concentrations being recorded near the open fire and lower concentration being recorded near the door and window apertures. The spatial variation in NO_2 concentrations was 1-12 ppb. In this case the area to the top left corner experienced a greater proportion of concentrations of 8 to 10ppb, than in the measurements taken at 300mm and 600mm vertically.

5.5.5 NO_2 concentrations 1500mm from the floor

9	9	9	9	9	12	11	11	9	7	6	6	5	4	3
9	9	9	8	8	8	11	10	10	7	5	4	4	3	3
7	7	8	9	8	8	9	9	9	7	5	4	4	4	3
7	7	8	7	7	8	9	8	9	8	3	3	2	3	3
5	5	6	5	4	4	6	7	8	5	3	2	3	3	3
3	3	3	4	4	5	3	3	4	3	3	2	2	3	3
1	3	4	5	3	4	3	4	6	4	3	3	3	2	3
2	3	5	4	5	4	3	3	3	3	3	3	2	3	4
2	3	3	4	3	3	4	3	3	3	3	3	2	3	4
4	5	4	4	3	3	4	3	3	3	3	3	2	4	4
4	4	5	4	3	4	4	4	3	3	3	3	2	4	4

fig. 5.5.5(i) NO_2 concentrations at 1500mm from the floor

Discussion

Figure 5.5.5(i) indicated the NO_2 concentrations measured in ppb across the floor of the room at a height of 1500mm. The table showed a spatial variation in NO_2 concentrations in the room with higher concentrations of 10-12ppb being recorded near the open fire and lower concentrations of 1-5ppb being recorded near the door and window apertures. The air flow model generally correlated to the NO_2 concentrations in the room measured at 1500mm vertically.

5.6 Airflow modelling compared with real-time NO_2 levels measured in house number 3

Figure 5.4.3(i) represents the airflow in part of house number 3. The room to the left of the model was the living room of the house and had displayed greater variation in concentrations of NO_2 between the monthly mean level and the real-time measured level, than the other houses surveyed. The air flow model indicated that the air flow around the window and door apertures was greater than the rest of the room, where there was little or no airflow in the corners and that the air flow between the hall doorway and opposite window provided air movement across the room.

The NO_2 real time monitoring indicated that there was an inverse correlation between the air flow and NO_2 concentration i.e. in areas of greater air flow, the NO_2 concentration was lower. This was particularly evident around the area of the open fire. As the distance away from the fire increased, the concentration of NO_2 decreased. However, this was also affected by the air flow, where the air flow was greater the concentration of NO_2 decreased. There was, therefore, a correlation between air flow and NO_2 concentration that was confounded by the open fire contributing to the NO_2 concentration in the room.

It was noted that the greatest NO_2 concentration of 13ppb adjacent to the open fire was at 300mm, i.e. the height at which the fuel was burning. The lowest concentrations of NO_2 of 1-2ppb were measured in the area in front of the openable window where the air flow was greater. The higher levels of NO_2 were measured in the top left hand corner of the room where levels of 8-10ppb were measured at each horizontal level.

When comparing the five levels that NO_2 concentrations were measured at, 900mm above the floor showed the strongest correlation with the air flow model.

It was noted that there was little spatial variation between the NO_2 concentrations measured at the five monitoring heights so in practical terms a single set of

measurements at respiration height should suffice for most risk assessment exercises. This is demonstrated graphically at figure 5.6(iii) where the mean variance by height was only 0.35ppb.

Statistical analysis

The detailed descriptive statistics for these calculations are found in Appendix 5.

ANOVA	df	F	P	R2
Regression	1	40.884	0.0001	0.8196
Residual	9			
Total	10			

table 5.6(i) Results of regression analysis between 'distance from wall (mm)'and 'mean NO_2 level (ppb)'

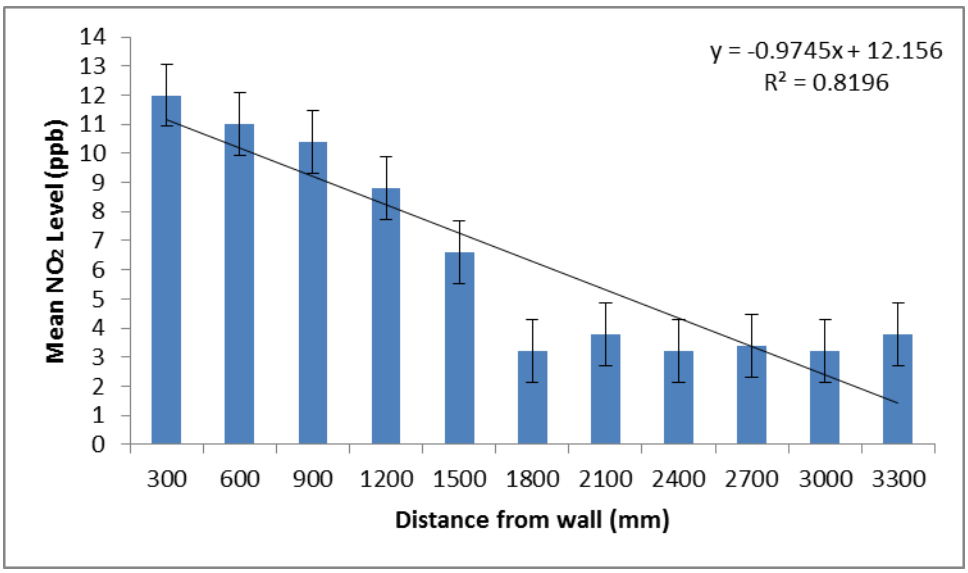


fig. 5.6(ii) Linear regression for relationship between 'distance from wall(mm)' and 'mean NO_2 (ppb)'

Table 5.6(i) provides statistical data for the linear relationship between the distance from the wall with the open fire and mean NO_2 concentrations at the transect line from the fire place.

The R^2 value of 0.8196 indicated that 81.96% variation in mean NO_2 concentration (ppb) could be statistically accounted for by the increasing distance from the wall (mm).

A p-value of 0.0001 showed a significant result for the relationship between NO_2 levels and the distance along the transect line from the open fire.

The F value of 40.884 concurs that the result was significant.

The ‘degree of freedom’ (df) showed a total of 10.

Figure 5.6(ii) demonstrated the linear relationship between the independent and dependent variables; mean NO_2 levels and the distance from the wall with the open fire and supports the data in table 5.6(i).

Evidently there was a significant statistical correlation between the NO_2 concentration and distance from the wall along the transect at the open fire i.e. there was a decrease in NO_2 concentration with increasing distance from the wall along the transect at the open fire. This indicated that there were variable levels of NO_2 in the room and therefore variable dispersion which could contribute to the development of micro environments.

Figure 5.6(iii) details the mean concentration for the various levels from the floor. It can be seen that the lowest mean concentration of NO_2 of 4.58ppb was found to be at 900mm from the floor. This would be the typical height of respiration for someone sitting down e.g. watching television, or working at a workstation.

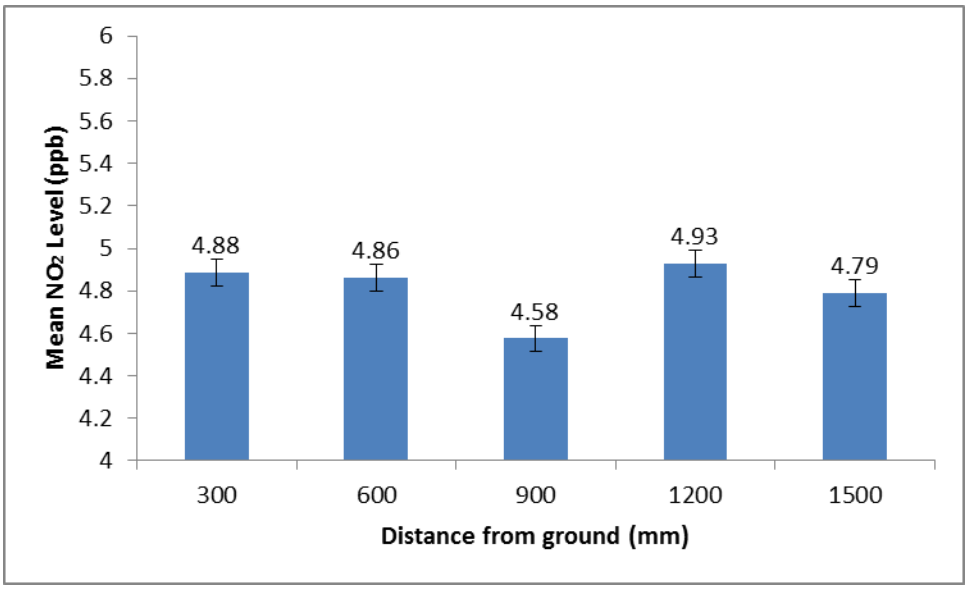


fig. 5.6(iii) Mean NO_2 levels at five distances from ground

5.7 Human exposure assessment

During this research it was possible to assess data for the mean monthly concentrations for NO_2 in ambient air and also those found in typical workplaces and homes in Guernsey.

From the literature it was revealed that human exposure assessment could be carried out by adding together the various exposures during the day to give the total daily exposure (Hetes, 1991; US EPA, 2012). This should be undertaken by measuring real-time exposure in each environment experienced by the case study. The following study could be used as a proxy, by using mean concentrations measured for each of the study locations for the multiple environment assessment of the various case studies.

The following examples were studies of one of the residents of houses 1, 2 and 3 and provide an overview of typical daily exposures in Guernsey during February 2012 and from which the hourly mean can be calculated.

5.7.1 Resident –house 1

12 midnight to 07.30	Sleeping bedroom	7.5 x 8.8ppb = 66ppb
07.30 - 08.30	Preparation for work – kitchen etc.	1 x 10ppb = 10ppb
08.30 -09.00	Cycled to work	0.5 x 22ppb = 11ppb
09.00 – 12.30	In office	3.5 x 3.8ppb = 13.3ppb
12.30- 13.30	Lunch time run	1 x 22ppb = 22ppb
13.30 – 17.30	In office	4 x 3.8ppb = 15.2ppb
17.30 – 18.00	Cycled home	0.5 x 22ppb = 11ppb
18.00 – 19.30	In house	1.5 x 10ppb = 15ppb
19.30 – 21.00	Football practice	1.5 x 22ppb = 33ppb
21.00 – 11.59	In house	3 x 10ppb = 30ppb
		Total daily exposure of NO_2 = 226.5ppb NO_2 hourly mean= 9.4ppb

table 5.7.1(i) NO_2 exposure assessment –resident house 1

Discussion

The NO_2 concentrations have been taken from the diffusion tube survey for house number 1, office number 1 and the ambient mean concentration for the area of St Peter Port where the house was located (College St - St James).

The total daily exposure of NO_2 of 226.5ppb was contributed to by ambient exposures during cycling and football activities and the rest by indoor exposures in the home and at work.

5.7.2 Resident – house 2

12 midnight to 07.00	Sleeping bedroom	7.0 x 5.3 ppb = 37.1ppb
07.00 - 08.00	Preparation for work – kitchen etc.	1 x 3.5 ppb = 3.5ppb
08.00 -08.30	Drove to work- dropped wife in SPP	0.5 x 28.10 ppb = 14.05ppb
08.30 – 13.00	In office	3.5 x 4.6 ppb = 16.1ppb
13.00- 14.00	Home for lunch (including drive there and back)	$^3/_4$ x 3.5 ppb = 2.625ppb ¼ x 28.10ppb =7.025ppb
14.00 – 17.00	In office	4 x 4.6 ppb = 18.4ppb
17.00 – 17.30	Drove home- collected wife from SPP	0.5 x 28.10 ppb = 14.05ppb
17.30 – 19.30	In house	1.5 x 3.5 ppb = 5.25ppb
19.30 – 22.45	Guests for dinner	$3^1/_4$ x 3.5 ppb = 29.75ppb
22.45 – 12.00 midnight	Off to bed	$1^1/_4$ x 5.3ppb = 6.625ppb
		Total daily exposure of NO_2 = 154.5ppb NO_2 hourly mean= 6.4ppb

table 5.7.2(i) NO_2 exposure assessment- resident house 2

Discussion

The NO_2 concentrations have been taken from the diffusion tube survey for house number 2, office number 2 and the ambient mean concentration for the Vale where the house was located (Vale Avenue).

The total daily exposure of NO_2 of 154.5ppb was contributed to by ambient exposures during travel to and from work and the rest by indoor exposures in the home and at work.

5.7.3 Resident – house 3

12 midnight to 07.30	Sleeping bedroom	7.5 x 3.3 ppb = 24.75ppb
07.30 - 08.30	Preparation for work – kitchen etc.	1 x 6.7 ppb = 6.7ppb
08.30 -09.00	Drove to work-	0.5 x 5.67 ppb = 2.83ppb
09.00 – 13.00	In office	4 x 4.6 ppb = 18.4ppb
13.00- 14.00	Office lunch	1 x 4.6 ppb = 4.6 ppb
14.00 – 18.00	In office	4 x 4.6 ppb = 18.4ppb
18.00 – 22.00	Drove home	4 x 5.67 ppb = 22.68ppb
22.00 – 12.00 midnight	Off to bed	2 x 3.3ppb = 6.6ppb
		Total daily exposure of NO_2 = 104.96ppb NO_2 hourly mean= 4.37ppb

table 5.7.3(i) NO_2 exposure assessment – resident house 3

Discussion

The NO_2 concentrations have been taken from the diffusion tube survey for house number 3, office number 2 and the ambient mean concentration for a proxy site at La Passee, which has a similar environment to Castel, where the house was located.

The total daily exposure of NO_2 of 104.96ppb was contributed to by ambient exposures during travel to and from work and the rest by indoor exposures in the home and at work.

From this study it can be seen that total daily exposure varied due to the spatial variation in daily experiences. In all case studies the hourly mean exposure concentration calculated was below the ambient mean for the area.

It was accepted that this study was limited as it did not use real-time measurement of NO_2 although it did identify a temporal and spatial variation in exposures due to individual experiences during a 24 hour period.

5.8 Conclusion

The studies in this chapter indicated that generally indoor NO_2 concentrations in home environments were lower than ambient levels. However, indoor combustion processes caused elevated levels of NO_2. In order to mitigate this effect, building design would be vital to ensure that open combustion processes were not located in living spaces to reduce NO_2 exposures.

The air flow modelling exercise was carried out using basic air flow modelling software, however, the results indicated spatial variations in the five locations modelled due to their various building layout and design. The positioning of doors and windows had an impact on indoor air flow.

The studies assessing the spatial distribution of NO_2 in house number 3 indicated that NO_2 existed in varying concentrations within the room and that, due to a combustion process and the position of doors and windows affecting air flow, low concentration micro environments had developed within the room. The study indicated that vertical spatial variation was minimal, 1-3ppb although horizontal spatial variation was 1-13ppb across the room. This suggested that further studies carried out should be at the height of respiration, rather than a series of measurements at different heights. This would

save considerable time and resources if such an exercise was undertaken during the risk assessment processes.

Hasselblad (1996) reported that long-term exposure to indoor mean levels NO_2 of 15ppb had an impact on the respiratory health of study subjects. If an indoor concentration 15ppb for NO_2 was used as a benchmark, then none of the houses studied during this research would be likely to have an impact on the respiratory health their residents. The same could be said for the workplaces studied, except for the catering kitchen where the NO_2 monthly mean concentration varied from 16.6ppb to 18.8ppb with a four monthly mean of 17.8ppb.

The studies in this chapter indicated that ambient air quality measurements can act as a proxy for human health as in most cases the indoor air quality measurements were lower than ambient. Thus the control of ambient air quality measurements will provide adequate protection for the respiratory health of the population. However, there were confounding factors, particularly the use of indoor open combustion processes that contributed to elevated concentrations. Where indoor open combustion processes were present, then a detailed exposure assessment would need to be carried out using real-time measurement and analysis.

It was noted that the exposure assessment of three office workers identified that one case study who lived near a major road, undertook outdoor exercise etc. had a higher daily exposure of NO_2 of 226.5ppb, and the other two other case studies whose daily activities where primarily indoors, revealed lower daily exposures of 154.5ppb and 104.96ppb. This outcome showed that living close to a busy road in Guernsey and spending time outdoors in that environment increased exposure to NO_2 and that the exposure could be more than double the exposure of someone living in a rural location.

Chapter 6

Scottish Environmental Health Officer survey

Overview

In 2011 the authorised officers and Environmental Health Officers from 13 out of 32 Scottish Local Authorities provided information on their perception of asthma in the workplace and their approach to risk assessment of workplaces. This chapter will explore the outcomes of the survey, including an analysis and evaluation of the participant's responses.

The survey questionnaire is attached in Appendix 6 along with the Microsoft Access database containing the responses.

6.1 Introduction

- To evaluate the role and approach of Environmental Health Officers in assessing the risks to pre-existing asthmatics in the indoor workplaces within their regulatory remit.

Environmental Health Officers (EHOs) based in local authorities have a regulatory role in undertaking the inspection of workplaces to ensure compliance with the Health and Safety at Work etc. Act 1974, and its subordinate legislation. This legislation is aimed at protecting employees and visitors entering workplaces from hazards and eliminating, or reducing to a minimum, any risks in those workplaces.

In addition, EHOs have a remit to inspect dwellings to ensure they are habitable and do not impact on the health and wellbeing of the occupants.

This combined with the air quality management role of local authority EHOs, means that these professionals are ideally placed to tackle the 'asthma epidemic' and to bring about real improvements in the indoor environments of homes, workplaces and outdoors, through a range of interventions, and by enforcement if necessary, to improve the health and wellbeing of asthmatics

Whilst the training and development of professional officers is regulated by 2 professional bodies in the UK (the Chartered Institute of Environmental Health (CIEH) and the Royal Environmental Health Institute of Scotland (REHIS)), the approach taken by individual officers once they are qualified is not known. A review of the literature revealed little evidence about the consistency or approach taken by EHOs. In 2007 a consultation document was circulated by REHIS which stated that 86% of Scottish EHOs were not involved in any form of work-based training and 34% said they had never had any work-based training after qualification (REHIS, 2007). In addition to employing EHOs, some local authorities employ other suitable qualified or experienced personnel who may be authorised to undertake certain regulatory functions, and are known as "authorised officers"

During the routine performance management of services, it is good practice for officers to be audited by their peers and supervisors to ensure a consistent approach is taken to their regulatory activity. A review of the literature revealed little evidence in this area so whether peer review happens routinely in local authorities is unknown.

This research, about the approach of local authority EHOs and authorised officers to risk assessment of asthmatics at work in Scotland, was unique and was undertaken in 2011. The questionnaire was designed using short easy to understand questions, with a series of multiple choice tick boxes for the answers. It was assumed that the participants were professional staff who should have a detailed understanding of the subject.

The questionnaire was designed to establish what EHOs and authorised officers thought about asthma and their approach to regulation of the premises within their remit that may have an impact on asthmatics. The participants were asked to respond on behalf of their local authority from their departmental perspective, rather than their own personal perspective, whenever possible.

The questionnaire was devised to evaluate the knowledge of EHOs and authorised officers about the key criteria impacting on asthmatic health status, such as humidity and temperature, specific environments known to cause sensitisation etc. This followed on from joint training that had been delivered to HSE inspectors and local authority EHOs as part of the HSC campaign to address asthma, dermatitis and asbestosis in the workplace. This training programme was undertaken in 2007 as part of the ‘Disease Reduction Programme’ (DRP) and the introduction of ‘themed’ inspections, although it was acknowledged that many officers did not attend the training offered due to travel and time constraints. Whether the training was cascaded in departments was unknown.

This survey was aimed at assessing some of the gaps in current knowledge about the activity of individual EHOs and authorised officers and their departments.

6.2 Analysis of the EHO survey responses

6.2.1 Participant details

Of the thirteen local authorities that responded, only 2 (15%) responses were from authorised officers, the rest were from EHOs. Of the 11 (85%) EHOs that responded, 2 (18%) were Health and Safety Managers and 2 (18%) were employed in specialist health and safety teams. The rest (64%) were generalist EHOs who undertook health and safety inspections as part of wider remit.

6.2.2. Operational partnership agreements

As part of the HSC campaign to address the DRP and themed inspection approach across local authorities and the HSE, operational partnership agreements were introduced. The responses to this question indicated that 9 (69%) local authorities out of the 13 participating authorities had operational partnership agreements in place.

6.2.3 Fit3 programme participation

All respondents agreed that their department was taking part in the Fit3 programme (100%).

6.2.4 Themed or topic based inspections

All respondents agreed that they were taking part in themed or topic based inspection programmes in an effort to prioritise workloads.

6.2.5 Effectiveness of themed inspections

12 out of 13 (92%) respondents agreed that they thought themed or topic based inspections were effective in their local authority area.

6.2.6 DRP topics as themed inspections

Participants were asked to state whether they would give priority to the three DRP topics during themed inspections. The responses were that all 13 (100%) agreed that they did prioritise dermatitis, 10 out of 13 (77%) prioritised asbestosis and only 7 out of 13 (54%) would prioritise asthma as detailed in figure 6.2.6(i).

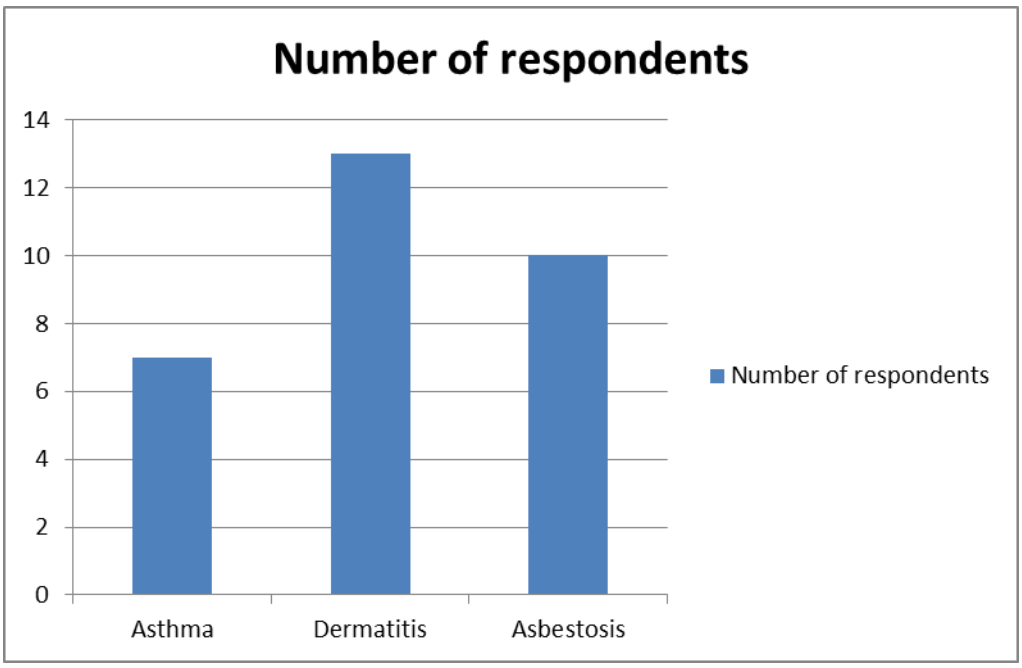


fig 6.2.6(i) Prioritisation of DRP topics

6.2.7 DRP priorities as part of the new OPA

8 out 13 (62%) respondents stated that they had continued to prioritise the DPR topics as part of their new OPA.

6.2.8 Knowledge to risk assess pre-existing asthmatics at work

Only 6 out of 13 (46%) respondents agreed that they had sufficient knowledge to undertake a thorough risk assessment of a pre-existing asthmatic in the workplace**.**

6.2.9 Environmental factors in workplaces impacting on pre-existing asthmatics

Participants were invited to respond to a list of environmental factors that may impact on asthmatics in workplaces. These included dust and fume, temperature and humidity as detailed in figure 6.2.9(i). All respondents (100%) agreed that dust and fume were important environmental factors when considering risk assessment of asthmatics at work. 8 out of 13 (62%) respondents thought that high humidity and low temperature were relevant factors, 7 out of 13 (54%) agreed that low humidity was an important risk

factor, 6 out of 13 (46%) agreed that high temperature was and important risk factor and 5 out of 13 (38%) respondents thought that steam was an import risk factor.

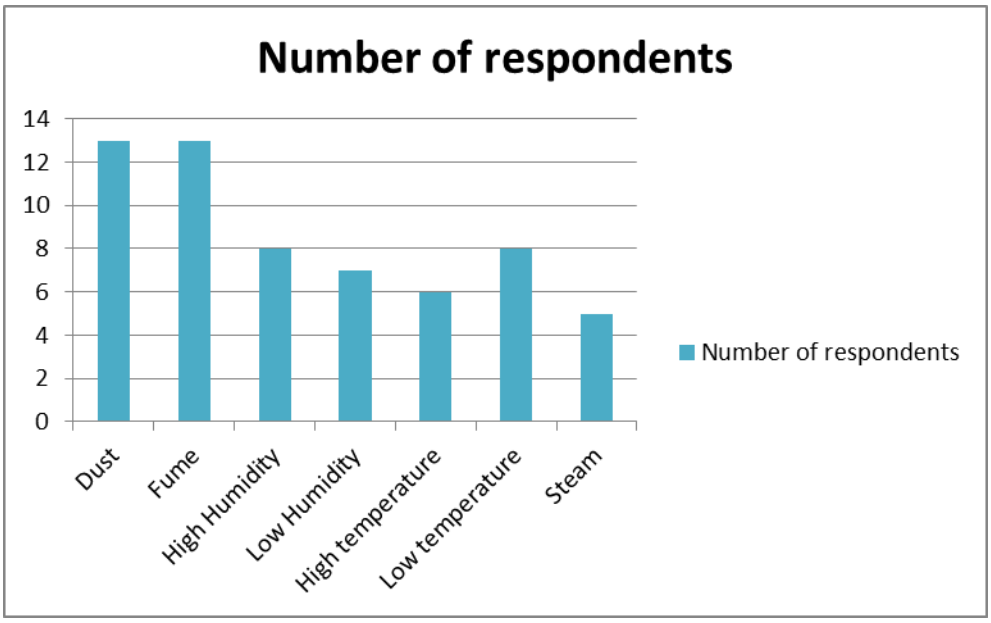


fig. 6.2.9(i) EHO responses to environmental impacts on pre-existing asthmatics

6.2.10 Hazards in workplaces affecting asthmatics

Participants were invited to consider a list of typical workplaces that they would inspect during their regulatory activities and identify the types of premises where they would consider asthma as part of the risk assessment process during an inspection. The work activities on the list were selected to provide a range of workplaces that would normally be inspected by local authority EHOs and should be familiar to the participants in the survey.

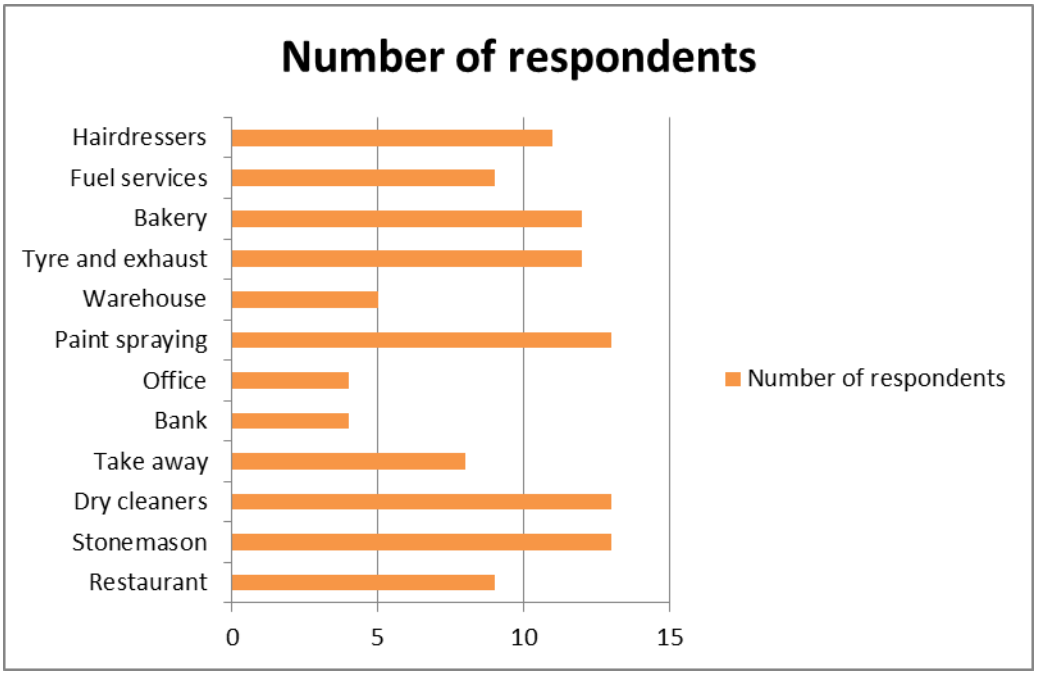


fig. 6.2.10(i) Workplaces with hazards that EHOs agreed may affect asthmatics

Figure 6.2.10(i) details the number of respondents who would consider asthma as part of a risk assessment during a premises inspection for the listed work activities. It was interesting to note that the majority of respondents (85-92%) considered premises where dusts and chemicals were used to be a higher priority for asthmatics than offices and banks (31% of responses), with warehouses (38% of responses) being of lower risk. The respondents indicated that they thought that take-away's (62% of responses) and restaurants (69% of responses) were of moderate risk to pre-existing asthmatics in the workplace.

Whilst there was space for the respondent to add notes or caveats, none of them did so.

6.2.11 Consideration of asthma when scoring risk

Following a health and safety inspection, the EHO will write a report of the inspection and undertake the health risk and safety risk scoring process which will determine the frequency of inspection for the premises. Only 4 of the respondents (30%) said they consider asthma as a health risk during the scoring process.

6.3 Conclusion

The survey of EHOs and authorised officers in 13 Scottish local authorities provided a unique insight into the processes being undertaken by this group of workplace regulators and this study had never been undertaken before. There was no literature or other evidence upon which to draw comparison or contrast in this context.

Assessment of the outcomes of the survey revealed that the participants were involved in national strategies and policies aimed at reducing workplace ill health and were aware of strategies such as the Fit3 Programme, Disease Reduction Programme and Operational Partnership Agreements. In addition, these policies were aimed at saving resources and prioritising workloads so would be an important tool for local authorities during times of diminishing resources.

There was, however, some concern about the perceived lack of knowledge of participants about asthma and risk management of asthma in the workplace. Whilst training on the three DRP topics had been delivered jointly to Local Authority staff and HSE inspectors in 2007, this may not have been cascaded to all staff.

Most of the respondents were familiar with a number of criteria impacting on the health of asthmatics, with a particular focus on dusty environments and those where chemicals were used. However, fewer respondents were concerned about workplace activities such as offices and banks.

It was disappointing to note that only 4 out of the 13 (30%) respondents would consider asthma when undertaking a risk assessment priority rating score of a workplace following inspection. This could have the effect that these workplaces would be inspected less frequently and therefore, there would be less opportunity for asthmatics to be effectively risk assessed and risk managed by regulatory activity. The literature revealed that as many as 1 in 10 people entering the workforce could be asthmatic (Asthma UK, 2008), then this could be an important omission in the risk assessment and risk management process for those individuals.

Chapter 7

Development of a risk management framework for pre-existing asthmatics at work

Overview

This chapter draws together the information from the foregoing chapters in establishing the key criteria for a risk management framework for pre-existing asthmatics in the workplace.

The research indicated that asthma was a widespread condition and that as many as 1 in 10 people in the UK entering the workforce could be pre-existing asthmatics and therefore risk management of these individuals in the workplace will prevent, or reduce to the minimum, health impacts and improve the health and wellbeing of those people. In addition there will be economic benefits in reducing days lost due to ill health and sickness absence.

Risk assessment and risk characterisation of allergen and pollution sources, concentrations and duration of exposure, building design and airflow, was included in the development of the risk management framework.

7.1 Introduction

The aim of this chapter was to achieve objectives 1 and 3 detailed in Chapter 1.

- To provide information for employees, employers and regulators about key risks to pre-existing asthmatics in the workplace.
- To create a simple risk management framework for asthma for employers, employees and regulators

This chapter is the culmination of this research. All of the evidence will be drawn together to establish a simple risk management framework that can be used by employees, employers and regulators to improve the risk management of asthmatics in the workplace. The framework could also be used for other environments and for total exposure assessment, with some minor modifications.

From the research, it was established that there were a range of complex, inter-relating issues that must be considered when carrying out a risk assessment of pre-existing asthmatics in the workplace, which then could lead on to improved risk management and better health for those individuals.

7.2 Inter-relating criteria

7.2.1 Ambient air quality

The research identified that ambient air quality data could be used as a proxy for indoor environments and the standards set in the NAQS provide a good proxy to protect human health in the absence of specific indoor air quality standards. The results of the research undertaken in chapter 5 indicated that ambient levels of NO_2 were similar or higher than that indoors, other than where there was an indoor open combustion appliance present. Therefore the monitoring of ambient air quality within the standards set in the NAQS is an important health protection measure.

7.2.2 Indoor pollution sources

Indoor sources of pollutants such as NO_2 from indoor combustion processes contribute to the concentrations indoors, which can be significantly higher than ambient levels. This was evidenced by the research undertaken in Chapter 5 showing that concentrations of NO_2 in residences and workplaces with indoor open combustion appliances was significantly higher than ambient concentrations (table 5.3.1(i)).

7.2.3 Building design

Indoor building design and layout also contributed to increased levels of pollutants found in micro-environments, especially in the corners of rooms and corridors. This was exacerbated by poor air flows and inadequate ventilation, especially during the winter months.

In addition, the literature confirmed that asthmatics were sensitive to temperature and humidity in buildings and indoor allergens, that were specific to the individual, were much more difficult to manage through interventions in the workplace (HSE, 2008). The research undertaken in Chapter 5 indicated that airflow and ventilations were affected by building design, such as the location of door and window openings,

7.2.4 Health Status of individual asthmatics

There were co-effects such as when the temperature was warmer, there may be elevated pollen levels and people at work open the windows so ambient conditions were more prevalent indoors. When the outside temperature was cooler, this was often associated with damper weather, closed windows and greater use of indoor combustion processes and heating systems (Price, 2007). The research in Chapter 3 provided evidence of seasonal variation in NO_2 concentrations and seasonal variation asthma patient hospital admissions.

7.2.5 Exposure to pollutants

The exposure was calculated as the concentration of the pollutant multiplied by the time of exposure in hours. For workplace exposures this would be the sum of those exposures in hours divided by 8 to determine the time weighted average. This could then be compared with HSE guidance EH 40 which provided information on occupation exposure limits for many work based chemicals, gases and sensitising agents to ensure exposure was minimised or prevented, although this guidance does not include NO_2.

7.2.6 Risk Assessment and Risk Management

Risk assessment was the process that drew together the assessment of exposure, health effects and health impacts (WHO 2002). Once completed, the risk assessment needed to be considered in context with and the measures (WHO 2002) to mitigate those impacts on the individual and how the impacts could be prevented or reduced to a minimum. This should be a continuous process which leads to effective risk management.

7.3 Draft risk management framework

Figure 7.3(i) draws all of these concepts together in a simple framework for pre-existing asthmatics at work. The intention of the framework was that it could be used by asthmatic employees, employers and regulators to assess risks to pre-existing asthmatics at work and to develop management interventions to effectively manage risk.

7.4 Risk management framework test and calibration

In order to test the risk management framework and to calibrate the draft version, it was tested by two asthmatics, one employer, one enforcement officer and three EHOs in Guernsey, who volunteered to undertake the process.

Each volunteer was given a printed copy of the draft risk management framework and asked to comment on the design and content. They were given brief information about the draft framework, i.e. it was developed to assist with the risk management of asthmatics at work but they were not given any specific detailed information about the research.

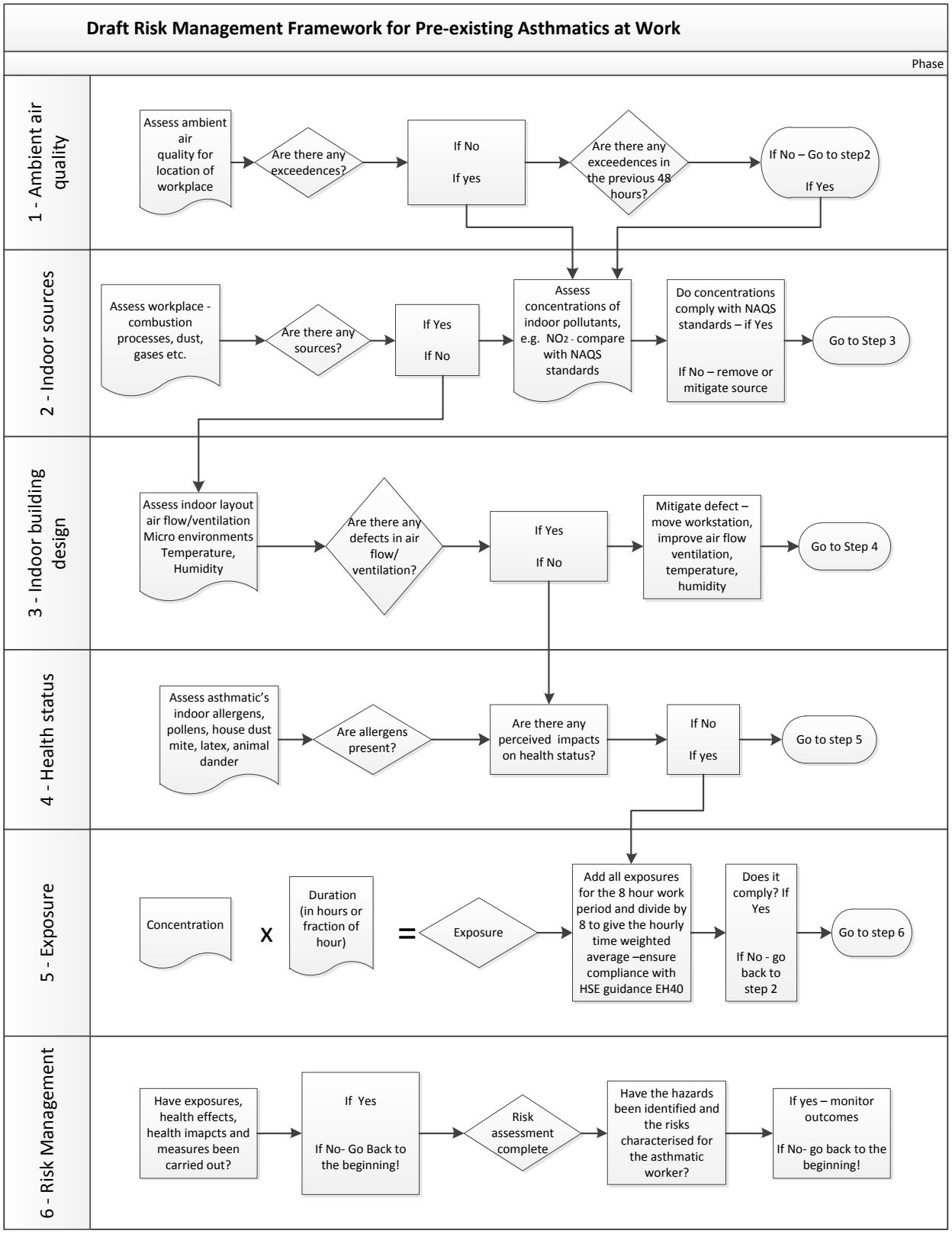


fig. 7.3(i) Draft risk management framework for pre-existing asthmatics at work

7.5 Outcomes of the test and calibration exercise

7.5.1 – Responses from asthmatic participants

Step 1- Ambient air quality. Both asthmatic respondents did not understand the term 'ambient air quality' and both respondents said they would not know where to get the information from about ambient air quality.

Both respondents queried the wording of the second step as they did not know what 'exceedences' were or how to get the information about standards.

Step 2-Indoor sources. Both respondents did not believe they would be able to assess indoor combustion processes or understand what indoor 'sources' were. They did not know what the NAQS was.

Step 3- Indoor building design. The respondents did not think they would be able to assess indoor building layout and air flow or humidity, although they did have access to a thermometer to check the temperature. They both agreed that they would be able to move their desks if they needed to improve the location of their workstation.

Step 4- Health status. One respondent assumed that this meant their own health but the other respondent thought this meant the general health of the people in their workplace. Both respondents queried why there was no 'yes/no' question in relation to the presence of allergens.

Step 5 – Exposure. One respondent was aware of HSE guidance- EH 40, but the other did not know about HSE guidance.

Step 6 – Risk management. Both respondents made no comments about this.

7.5.2 – Responses from an employer

Step 1 – Ambient air quality. The respondent queried whether this was 'outdoor' or 'indoor' air quality and commented that he would not have access to this information.

Step 2- Indoor sources. The respondent made no comments.

Step3 – Indoor building design. The respondent made no comments.

Step 4 – Health status. The respondent queried why there was no 'yes/no' decision box after the question about the presence of allergens.

Step5 – Exposure. The respondent made no comments.

Step 6 – Risk management. The respondent made no comments.

General comment – the respondent said that this would be a useful tool if it was amended.

7.5.3 – Responses from 3 EHOs and an Enforcement Officer

Step 1- Ambient air quality

EHO1 responded that Step 1 could be streamlined by merging the questions about the ambient air quality and the question about the air quality in the previous 48 hours.

EHOs 2 and 3 said they assumed that 'exceedences' referred to the NAQS standards and would clarify that question. They also commented that they thought that the risk management framework 'steps' should be termed 'stages', as each 'stage' had a number of 'steps'.

The enforcement officer responded that 'exceedence' should refer to the standards and queried whether the question box relating to the exceedences in the previous 48 hours could be relocated.

Step 2 – Indoor sources

EHO1 suggested that if the source of pollution was mitigated or removed, the assessor should return to Step 1 to check that the measures were effective.

EHOs 2 and 3 responded that they would want more clarity on the type of 'sources'.

The enforcement officer made no comments.

Step3 – Indoor building design

EHO1 queried whether this was already described elsewhere and queried what ‘defects’ meant. In addition, the direction of the arrow from the ‘yes/no’ decision box to step 4 would mean that the assessment of allergens would not take place so should be missed out.

EHOs 2 and 3 responded that they wanted to know which standard the assessment of air flow, ventilation, temperature, humidity etc. would be measured against.

The enforcement officer made no comment.

Step 4 – Health status.

EHO1 responded that there should be a ‘yes/no’ decision box after the question about the presence of allergens. In addition the direction arrow from the ‘yes/no’ decision box would prevent exposure assessment in step 5 so should be missed out. This responded queried whether if there were no pollutants could allergens be ignored.

EHOs 2 and 3 queried whether this question was about the individuals perception of their health or their medical assessment.

The enforcement officer made no comment.

Step 5 – Exposure

EHO1 had already commented that directional arrows needed to be realigned.

EHOs 2 and 3 commented that the first box should identify the concentration ‘of what’?

The enforcement officer made no comment.

Step 6 – Risk management

EHO1 commented that if there was no perceived health effect and that if all of the risk assessment characterisation steps had been completed, there was no need for steps 5 and 6.

EHOs 2 and 3 commented that if the exposures, health effects, health impacts and measured had not been carried out and commented that the assessor should go back to Step 2 rather than the beginning because they would not normally consider ambient air quality during a health and safety inspection.

The enforcement office thought the process should be completed if the risk assessment was completed.

7.6 Calibration of the draft risk management framework

It was evident from the comments made by the asthmatic participants that the draft risk management framework contained technical terms that were not understood and was too complicated for widespread use. They would not have access to the technical data need to complete an assessment on their own.

The employer had a reasonable grasp of the technical terms used but was not able to source some of the information and data that would be required.

The EHOs and enforcement officer who participated had a good knowledge of the technical terms used had a better understanding of the subject matter involved. This was reassuring as this group of participants are involved in health and safety matters on a routine basis.

Therefore, following this calibration exercise, the draft risk management framework was reformatted and validated for use by regulatory staff involved in inspection of workplaces and can be found at figure 7.6(i).

In order to complete the objectives of this thesis, a simpler information tool was developed for use by asthmatics in Guernsey and can be found at figure 7.6(ii). This was formatted as a three-fold information leaflet for easy use and display at GP practices, medical information centres etc. The research indicated that total exposure assessment was important for the management of asthmatics and therefore all factors affecting their health and well-being needs to be considered. This leaflet provides a novel resource for asthmatics because, for the first time, it combines all of the factors affecting asthmatics, the ambient environment, their health status and the indoor environment, both at home and at work, and so this approach to risk management of asthmatics is unique.

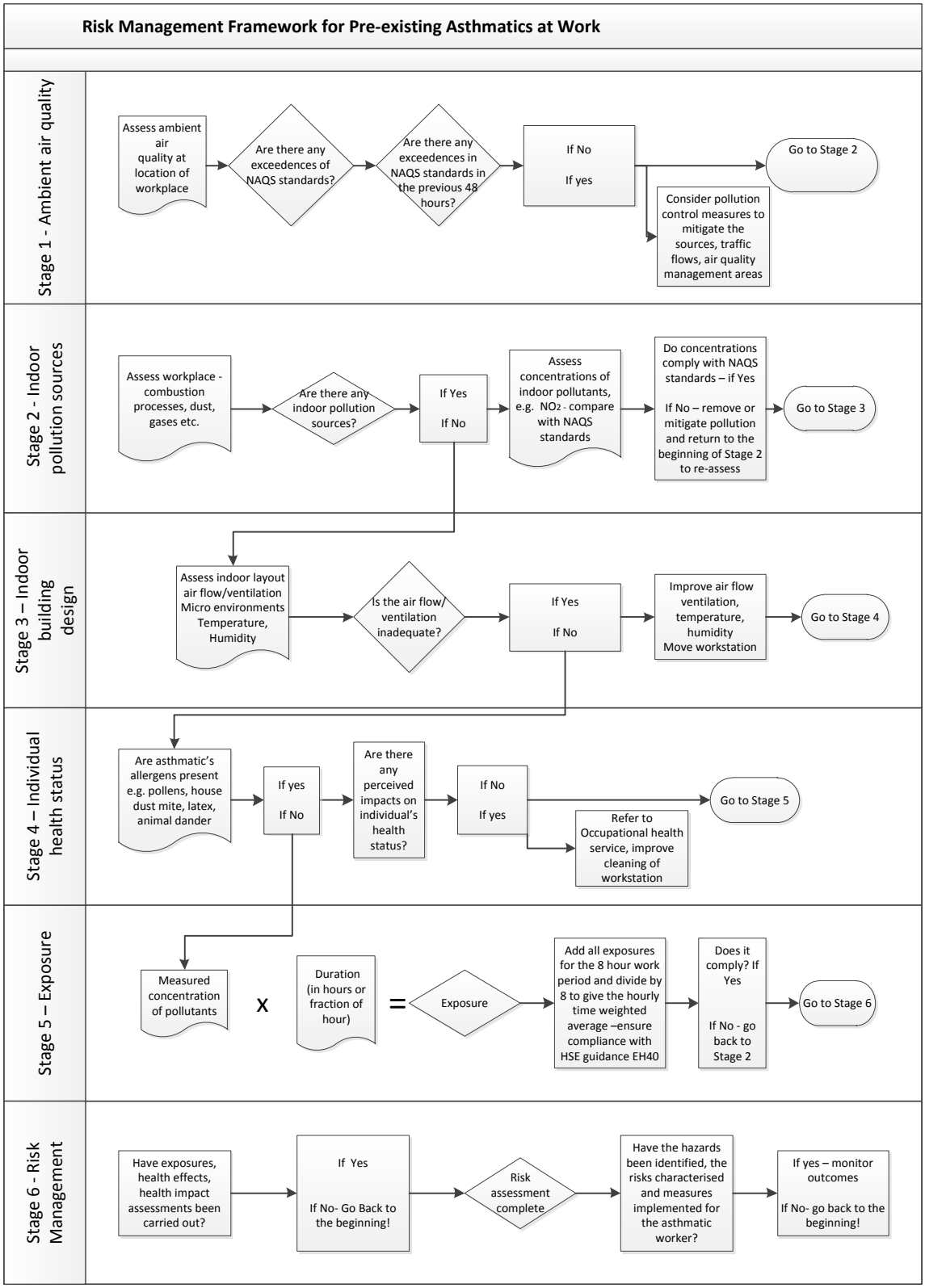
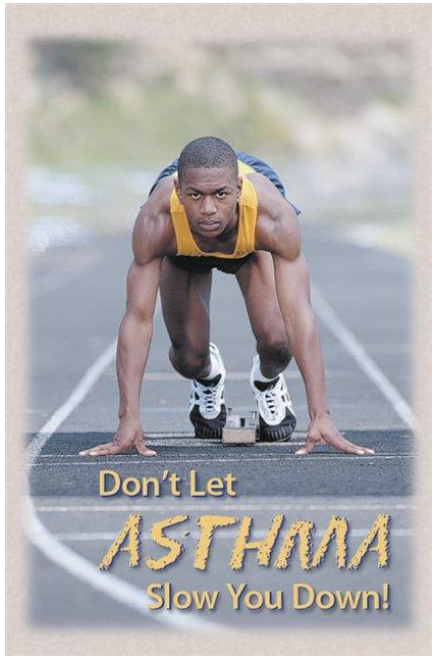


fig. 7.6(i) – Regulator's risk management framework for pre-existing asthmatics at work

fig. 7.6 (ii)



Contacts:-

Office of Environmental Health and Pollution Regulation

Tel: 711161

Health and Safety Executive

Tel: 234567

Ten Top Tips to improve your asthma in Guernsey

Managing your Asthma

People with asthma can be exposed to a range of allergens that trigger asthmatic episodes at home, at work, and out of doors.

Your GP will help you to manage the symptoms, but there are a number of simple things you can do to make your life easier.

Ten Top Tips

The Environment

Outdoor air pollution can cause your asthma to get worse, especially if your asthma has already been triggered.

1 Check air pollution levels near your home and your workplace with the Office of Environmental Health and Pollution Regulation to see if you are at risk.

2 Avoid walking along busy roads during rush hour to prevent exposures to traffic pollution.

3 Avoid walking in areas near dusty processes like building work, cement mixing, wood working etc.

At home

4 Make sure you have good air flow and ventilation in the rooms you use, especially where you sit for a long time or sleep.

5 Avoid open burning appliances such as open coal or wood fires, paraffin heaters, gas cookers and boilers unless they are well ventilated or have sealed flues.

6 Make sure your home is regularly ‘wet’ dusted, vacuumed and steam cleaned, especially if you are allergic to house dust mites or animals.

At work

7 Make sure there is good air flow and ventilation around your workstation.

8 If possible, avoid sitting in corners away from openable windows or doorways.

9 Check the temperature of your workplace, ideally it should be above16^{o}C for comfort.

10 If you have regular dry eyes, nose or throat, check the humidity in your workplace. You may need a health and safety specialist to assist you to do this. Often plants in the area can improve the humidity, as long as they don’t produce pollens.

Do Make sure your employer knows you have asthma so that a proper risk assessment can be carried out and measures put in place to reduce the risk of you having an asthma attack.

Chapter 8

Conclusions

Overview

This chapter draws together and presents the conclusions in relation to the aims and objectives of this research and summaries the key findings that have been identified. This chapter will include a summary of conclusions on the novel aspects of the research and what has been learned that can contribute to the evidence base in this field.

8.1 Introduction

This research project was aimed at contributing to the evidence base and understanding about pre-existing asthmatics and the impacts on their health and wellbeing at work and exacerbations from environmental stressors.

The research aimed to provide an understanding of the relationship between ambient and indoor air quality and the influences of building design, air flow and ventilation on indoor pollutant concentrations. Indoor micro environments were examined in this context with human exposure assessment.

The assessment of NO_2 was selected specifically for the various studies because it was the only ambient pollutant in Guernsey that exceeded the NAQS standards.

The aims of this research were:-

- To evaluate the factors that impact on the health of asthmatics at work so that a risk management framework can be developed for use by employees, employers and regulators.

- To examine whether ambient air quality is a good proxy for impacts on the health of asthmatics by examination of temporal and spatial variations in hospital admissions in Guernsey, for patients with asthma during 2008-2012 in comparison with ambient air quality data for NO_2.

- To evaluate multiple exposures and how multiple exposure assessment can be used in conjunction with the ambient air quality measurements to identify spatial and temporal variations.

- To identify whether micro-environments for NO_2 exist in buildings and whether building design and layout play a role in exposure assessment.

- To evaluate the role and approach of Environmental Health Officers in assessing the risks to pre-existing asthmatics in the indoor workplaces within their regulatory remit.

- To assess whether exposure to NO_2 is a necessary characteristic in risk assessment of pre-existing asthmatics in indoor workplaces.

In this thesis the outcomes from the various studies were described and discussed and a summary of conclusions follows.

8.2 Summary of conclusions

Chapter 3- Assessment of spatial and temporal data for hospital admissions for asthma patients in comparison with NO_2 levels in Guernsey 2008-2012

The research outlined in Chapter 3 was aimed at assessing whether there were influences from air pollution on the health of asthmatics in Guernsey, with specific interest in variation in NO_2 concentrations. Studies undertaken over a number of years by researchers including Hasselblad et al (1992), Tunnicliffe et al (1994), Linaker et al, (2002), Chauhan et al, (2003), Price, (2007), have indicated that spatial and temporal variation in NO_2 concentrations have an impact on the health and wellbeing of asthmatics and admissions to hospital.

The analysis and evaluation of the research data for this thesis identified spatial and temporal variation in NO_2 levels in Guernsey. This research had never been undertaken before in Guernsey and presented new and unique information which contributed to a better understanding of the local situation and the evidence base. The research showed that NO_2 levels in Fountain Street, Bulwer Avenue and Vale Avenue in Guernsey exceeded NAQS standards and was a cause for concern that required further study, assessment and evaluation. The research examined the patient hospital admission

numbers for asthma over a five year period (2008-2012) and the visual assessment of the data, graphs etc. indicated that there was spatial and temporal correlation between increases in hospital admissions for asthma when concentrations of NO_2 increased.

However, the statistical analysis of the data calculated no significant linear correlation other than for 2011 when there was limited statistical correlation.

The study also included an assessment of peak NO_2 concentrations and the lag-time of 48 hours for the dates of all hospital admissions during the study period. This method was based on research undertaken by Rusznak et al (1996) where symptoms of asthma were found to present up to 48 hours after exposure to elevated levels of NO_2. The study undertaken for this thesis substantiated the findings that a lag-time of up to 48 hours is an important factor in asthma patient assessment.

As with all population health studies, the size of the study cohort is important, the larger the cohort the more significant and reliable the findings (Farmer et al, 2004) and it was acknowledged that the data sets in this research were small and the outcomes of the statistical assessment should, therefore, be considered with caution.

Chapter 4 - Asthma patient study

The patient study in Guernsey provided a unique insight into the complexity of asthma. Initially there were challenges in engaging participants in such a study and gathering the survey returns for analysis. On the third attempt at this area of the research, in November 2013, five participants were fully engaged in the process and completed their daily diary survey forms for that month. The data was processed and compared with the real-time monitoring results for NO_2 that were available. One of the issues that arose during this study was that the real-time analysers deployed by the OEHPR were occasionally ‘off-line’ so the data set was incomplete and this made data comparison difficult. There were two important results that indicated the temporal correlation between asthma symptoms experienced by the participants and elevated ambient NO_2

concentrations from 2nd-11th and 23rd-29th November, 2013 when 4 out 5 participants experience wheeze or tightness of the chest concurrently.

The participants thought that colds and influenza-like viral illness caused their asthma symptoms to worsen. Viral infection was also identified as having a co-effect with asthma symptoms by Nicholson et al in 1993.

The participants provided some qualitative information about their condition although all were generally unclear about any environmental factors that made the asthma symptoms worsen. It was interesting to note that none of the participants considered air pollution in Guernsey to have an impact on worsening their asthma. One participant did report that living in London, however, had made their asthma worse during the time they lived there, so there was some level of subconscious consideration that air pollution had an adverse impact on their asthma symptoms.

Chapter 5 - Indoor NO_2 levels and exposure assessment

An area of focus for this research concerned the use of ambient air pollution data in making policy decisions about population health and the ensuing interventions that had been developed in the UK NAQS. The research aimed to test the hypothesis that ambient air quality is a proxy for human health by undertaking a series of tests to examine indoor exposures to NO_2 in comparison with those outdoors. Following international guidance on NO_2 monitoring (AEA, 2008; ESCAPE, 2010), three residential and four workplace locations were selected for the research. The sites reflected a variety of building types, design and layout, and the workplaces reflected the typical Guernsey workplaces that would not normally contribute to occupational asthma. The security of the sampling equipment was an important consideration when choosing sites.

The monitoring undertaken using diffusion tubes was later validated with a real-time gas analyser. It was revealed that NO_2 monthly mean concentrations in homes were

generally lower than ambient NO_2 monthly mean concentrations at the three residential study locations in Guernsey.

The real-time measurements of NO_2 concentrations taken for validation purposes in the three houses, showed that higher levels of NO_2 were measured in some rooms and therefore any detailed assessment would require real-time analysis of NO_2 concentrations. The research should not rely solely on the use of monthly mean measurements using diffusion tubes.

The measurement of NO_2 monthly mean concentrations in the four workplaces showed that two offices and a reprographics print room were similar or lower than ambient NO_2 monthly mean concentrations. However, the NO_2 monthly mean concentrations measured in a catering kitchen were higher than ambient NO_2 monthly mean concentrations due to the location of a range of open gas cookers and hobs.

The real-time measurements of NO_2 concentrations in the workplace study locations showed higher levels than the monthly mean concentrations measured by diffusion tube and the catering kitchen real-time measurements showed significantly higher NO_2 concentrations than the monthly means measured at the same location. The real-time measurements were recorded during the working day. The monthly mean sample results would have the effect of averaging the concentrations experience during the activities in the daytime and vacant use during the night-time, when there was no activity.

The indoor air flow modelling exercise indicated that building design and layout were important factors for good air flow and ventilation to ensure the dispersion of NO_2 in indoor environments. The location of window and door openings made a significant impact on the air flow in the rooms studied and this indicated that indoor micro-environments existed in the rooms studied. The levels of NO_2 in the rooms were generally below ambient NO_2 levels, therefore substantiating the use of ambient air quality monitoring and standards as a proxy for human health (UK NAQS).

However, in the room with an open combustion appliance (i.e. an open coal fire) it was recorded that the NO_2 concentrations were higher than ambient NO_2 concentrations, the closer to the open fire the higher the level of exposure to NO_2.

Although the research set out to examine the impact of indoor air quality on the health of pre-existing asthmatics at work, it was evident that total exposure assessment was an important factor in assessing the exposure of working individuals.

In order to test individual exposure assessment further, the three residential case studies and four workplace case studies in Guernsey provided unique data on the spatial variation in exposure due to location of residence, proximity of residence to busy traffic routes, workplace exposures and individual recreational exposures. It was concluded from this study that a person living in St Peter Port, who cycled to work and enjoyed recreational sports outdoors in the evening could have double the exposure to NO_2 than a person who lived in Castel parish, who drove to work and stayed indoors or went outside there during the evening. It was acknowledged that the daily exposures of all three case studies was low but that traffic emissions were a major contributing factor to NO_2 exposure in Guernsey. These results reflect earlier work by e.g. Gauderman et al (2005), Zhou et al (2008) and Karner et al (2010).

Chapter 6 - Scottish Environmental Health Officer Survey

There had been virtually no research done about the role and function of local authority EHOs in relation to their ability to influence the symptoms of asthma and improved health outcomes for asthmatics. This research programme started in Scotland and so this research focused on the staff employed in Scottish local authorities. Dating back to the nineteenth century, EHOs (and their predecessors) have been involved in the assessment of risk to health from environmental stressors (REHIS, 2007) and in delivering on national policy and strategic interventions at local level, so this cohort was selected for this research.

This was a novel area of research and the survey involved responses from 13 out the 32 Scottish local authorities and was undertaken in 2011. A survey was undertaken by email and the outcomes were inputted into a Microsoft Access database. This was a quick and effective method for gathering and analysing the data recorded.

From the analysis of the data it was concluded that all of the respondents were aware of national strategies and government initiatives and had implemented national programmes and policies in their local authority's work activities.

However, there was a perceived lack of knowledge about the risk assessment and risk management of asthma at work, raising concerns over the ability to undertake a thorough risk assessment encompassing all of the factors affecting an asthmatic at work, such as ambient air quality, indoor air quality, allergen exposure and the measures need to bring about improvement.

Although joint training about asthma at work had been delivered for HSE inspectors and local authority staff, this may not have been cascaded to all staff authorised under the Health and Safety at Work etc. Act 1974, resulting in reduced knowledge by some officials on risk assessment for asthmatics in the workplace.

Respondents were aware of the environmental impacts on asthmatics at work although focused on dusty workplace environments and workplaces using chemicals, with less emphasis or assessment of workplaces such as offices, restaurants, banks etc.

Following inspection of workplaces, the frequency of the next inspection was determined by a scoring method and only 4 of the 13 respondents said they would consider asthma as a health risk factor during the scoring process.

It was concluded that there may be the potential for workplaces where asthmatics are employed to be less frequently inspected and, therefore, the risk assessment and risk management of those asthmatics could be inadequate. It can be assumed that 1 in 10 people at work could be asthmatic (Asthma UK, 2008) and therefore this poses a significant risk for health and safety at work. This study identified a training and

development need for all local authority staff engaged in workplace inspection programmes.

Chapter 7 - Development of a risk management framework for pre-existing asthmatics at work

The conclusions of all of the aspects of the research were brought together to develop a draft risk management framework for asthmatics at work, with the intention that this could be used by asthmatic employees, their employer and regulators of workplaces. This research showed the detailed complexity of the factors involved in the risk assessment of asthma which were essential for effective risk management.

The evidence from the areas of study highlighted the importance of considering a range of factors that need to be assessed in combination, and not in isolation, if the risk assessment is to be effective and meaningful and bring about improvement in the health of asthmatics at work.

The draft framework calibration exercise involving two asthmatic workers, an employee, 3 EHOs and an enforcement officer, concluded that, due to the complexity of the subject, the regulatory staff needed a detailed framework to ensure all factors were taken into account during compliance inspections and priority risk rating and scoring processes.

The employer, whilst having a good understanding about risk assessment, felt that he would be unable to use the draft framework because, at that time, he would not have access to the necessary information in Guernsey, especially data about ambient air quality.

It was concluded that the risk management framework, which was calibrated, reformatted and validated, would be suitable for use by regulators only due to the range of technical knowledge and data required to complete the framework.

The outputs from the research about indoor air pollution, ambient air pollution, workplace and residential exposures, were used to create a simple information leaflet for use by asthma sufferers to assist them in managing their own condition in Guernsey.

Chapter 9

Appraisal and Recommendations

Overview

This chapter examines the research that has been undertaken and provides a critical evaluation of the research, its strengths and weaknesses and the gaps in the evidence that have arisen.

This chapter identifies areas for further work to fill the gaps and considers opportunities for improving the research should such a study be carried out again.

9.1 Appraisal and recommendations for further research

This project provided a range of new evidence about NO_2 and the spatial and temporal variations in concentrations in Guernsey and the impacts of NO_2 on the health and wellbeing of people with asthma.

Whilst air quality in Guernsey is generally very good due to the lack of industrial processes and its proximity to the confluence of the Atlantic Ocean and the English Channel and prevailing winds, the NAQS objectives and standards for NO_2 are regularly exceeded at some monitoring locations due to traffic emissions and so NO_2 concentrations in Guernsey were selected as a focus of the research.

Ambient air quality parameters have been routinely monitored in Guernsey for about twenty years, using a range of different equipment and methodology (States of Guernsey, 2010). This meant that temporal comparison of data trends over long periods of time was very difficult due the use of different equipment used and methods for data collection and presentation. The diffusion tube surveys had been varied considerably over the years, being deployed at over thirty sites, although at only ten to twelve sites at any given time.

The years from 2008 to 2012 showed some consistency and were selected as the study years for the project. However, whilst diffusion tube survey data was available, the real-time monitoring of NO_2 has only taken place for the last four years and even then the data was intermittent due to equipment breakdown or loss of computer connectivity and disconnection of one of the stations. Further work will need to be undertaken by the OEHPR to ensure consistency of data collection and presentation if there is to be a meaningful assessment of air quality monitoring data. In addition, the communication systems deployed to download real-time monitoring data failed on a number of occasions so work should be undertaken to improve data collection and storage.

Further research is required to identify methods for providing air pollution information to the public so that self-assessment of risk can be carried out to ensure personal health.

The asthma hospital admissions data was provided by the Clinical Coding team at the Princess Elizabeth Hospital, and provided useful information on acute exacerbations of the condition which required hospitalisation. However, professional conversations revealed that asthma was widespread in the community and less acute episodes were managed through the three GP practices on island. However, the total numbers were unknown due to disparate patient data collection systems. This issue was a matter that should be addressed urgently if the States of Guernsey was to undertake rigorous assessment of population health including asthma.

Admissions to hospital data for adults were likely to be confounded by the cost of private health charges at the Accident and Emergency admission stage and primary care costs, so the admission rate for adults was likely to be lower than a comparable location in the UK where GP consultations and admissions to hospital were free. The rate of admissions for young children was likely to be more accurate as most parents would pay for the health care treatment needed for their small child. Further research should be undertaken to assess the impact on asthma of the Guernsey health and social care system and the charging structures currently employed. The health care review in Guernsey, currently underway, will try to address general health inequalities created by the cost of health care.

The research relied on the use of expensive real-time monitoring equipment provided by the OEHPR, when it was available. The equipment could not be deployed and left unsupervised, so required continual supervision during the studies, some of which took many hours to complete. Availability of the Gray Wolf gas detector for the studies was also affected by its calibration requirements. It needed to be calibrated frequently and this was undertaken by a laboratory in the USA so the equipment was off island for periods of time on a routine basis.

The assessment of data presented in graphs provided trend lines of parameters such as NO_2 mean concentrations and hospital admissions and appeared to provide reasonable confidence in correlation, however statistical methods calculated using Microsoft Excel software indicated no significant linear correlation.

Due to the very small numbers involved such data assessment and statistical calculations for linear correlation of data should be reviewed with caution. Research involving larger data sets and cohorts will be needed to provide clear statistical information for further research projects.

In addition the correlation between NO_2 levels in St Peter Port and asthma prevalence in St Peter Port will be worthy of further research due to the traffic congestion and NO_2 levels exceeded there.

The surveys conducted were unique and have provided some new and interesting information. As with many such studies, the information provided has opened up avenues for further study. The literature review provided very little information about the role and function of EHOs in local authorities and how they conduct their regulatory activities. The EHO survey provided new evidence about compliance inspections and the risk assessment of asthmatics at work in Scotland. The training and development of EHOs and authorised officers in Scotland was sporadic and further work is needed if the risk assessment and risk management process is to be improved and therefore result in improved outcomes for asthmatics. These issues will be raised with the professional bodies in the UK to ensure additional training and development on risk assessment of asthma in the workplace is undertaken to address this issue (REHIS in Scotland and the Chartered Institute of Environmental Health in England/Wales/Northern Ireland).

There was a clear disconnect between EHOs in local authorities and health care professionals in the NHS and this should be addressed immediately to ensure that important environment factors, such as ambient air quality, hygiene of buildings and workplace compliance contribute to the asthma health improvement agenda, currently led by clinicians in the NHS.

The culmination of this project was to develop a risk management framework for use by employees, employers and regulators. It was acknowledged that the risk assessment and risk management of asthmatics at work was a very complex and technical topic and to achieve a simple tool to suit all needs was ‘over ambitious’ and had to be re-thought. The resulting asthma risk management framework for regulators and the information

leaflet have fulfilled the original aim but further work will be required to implement the use of the framework and improve information in this area.

It was clear from the work undertaken in this thesis that there needs to a greater understanding and more education at all levels about this widespread and debilitating condition if improved outcomes for asthmatics are to be achieved.

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APPENDICES

Appendix 1

A1.1 Strategy for Literature Review

A1.2 Literature scoring table

A1.3 UK Air Quality Objectives 2007

A1.4 Air Quality Standards Regulations 2010 - limit values

A1.5 Air Quality Standards Regulations 2010 – target values

A1.1 Strategy for Literature Review

Indoor environment and the impact on the health of pre-existing asthmatics at work – the development of a risk management framework.

Introduction
There are a number of requirements for developing a strategy for a literature review.

It is important to decide on the research question and to identify the key areas for literature search.

The sources of literature for the research need to be identified along with the strength of the evidence and the benefits and risks associated with each source. This will include texts and statistical data from primary and secondary sources used to back up critical evaluation and argument.

It is essential that the research focuses on the question.

For most literature reviews associated with PhD research programmes, a timetable needs to be drawn up and agreed.

Hierarchy of evidence

In public health, the 'hierarchy of evidence' is a system used to prioritise evidence from primary research i.e. studies based on experimentation or observation. Such studies are ranked, the higher the ranking the more accurate the evidence is likely to be.
systematic reviews and meta-analyses (the most important)
randomised controlled trials
cohort studies
case-control studies
cross-sectional surveys
case reports
expert opinion
anecdotal (of little or no importance)

Secondary sources bring together numerous pieces of published research into a single source. Many Government papers are secondary sources.

Inclusion/Exclusion of evidence

Based on the hierarchy of evidence, the criteria used to include or exclude evidence will be based around the key areas of the research question.

The question for the literature review:-

"Do indoor environments in workplaces such as shops, offices, catering premises, have any impacts on the health of pre-existing asthmatics, and are hazards addressed by current risk management interventions?"

The research covers three significant areas:-
Indoor air quality of work places
Impacts on pre-existing asthmatics
Risk management

1 Indoor air quality of work places

The outline of the research states that a large body or research exists for workplace environments that are known to cause asthma such as bakeries, wood working operations.
This study will examine indoor air quality and identify the parameters that impact on asthmatics.

2 Impacts on pre-existing asthmatics

The research will focus on 'normal' indoor work environments not currently associated with asthma e.g. offices, shops, food outlets.

3 Risk management

Whilst there is a duty on employers to assess the risks in their workplace and to document significant findings, many employers are unaware that they employ pre-existing asthmatics.
During routine inspections of workplaces, EHOs and HSE Inspectors undertake risk assessment and apply a framework to prioritise hazards and level of risk posed.

In 2005 the HSE introduced the 'Disease Reduction Programme' (DRP) which required themed, or 'topic-based' inspections of workplaces around the three high priority areas-asbestos, dermatitis and asthma.
The research will examine the application of DRP in respect of asthma.

Key words

Journal article searches will focus on the following key words and phrases:-

Indoor air quality asthma asthma at work air pollution
risk assessment risk management nitrogen dioxide and asthma

Evidence tables

Tables will be created for each of the key phrases, and sources will be logged throughout the review.
E.g.

Source	Hierarchy of evidence	Strength of evidence
Chauhan AJ, Inskip HM, Linklater CH et al, 2003: Personal exposure to nitrogen dioxide (NO_2) and the severity of virus-induced asthma in children. The Lancet 361:1939-1944	Primary 1	Strong

Strength of Evidence

Literature will be prioritized by the strength of the evidence found.

Strong	Conclusions supported by at least 2 peer-reviewed studies, primary sources with high quality or good systematic review
Moderate	Conclusions supported by 1study with good systematic review or 2 studies of medium quality review
Limited	Conclusions supported by 2 medium quality of systematic review
Insufficient	No studies meet the criteria
Contradictory	2 studies where contradiction found

Sources for the Literature Review

Source	Risk/Benefit
Professional journals	Peer reviewed articles Topic specific
Conference papers	Up-to-date Not yet published May/may not be peer reviewed
Research theses	Access Availability
Books	Published Peer reviewed May become out-of-date
Government Bodies - research papers	Expert input Published Peer reviewed Accurate
National/ International Working Groups	Expert opinion Papers published
Internet	Accuracy of source Date May/may not be peer reviewed World wide access to data
Newspapers	Up-to-date Problem with accuracy

Referencing System

The Harvard referencing system will be used to identify the sources of the evidence, quotation etc.
In the body of the text a source will appear as - Bloggs (2007).

All sources will be listed in the bibliography at the end of the literature review. Book and journal references will be arranged in alphabetical order by the first authors name and then chronologically e.g.

Bloggs, A., 2007. *Air quality and asthma*. New York : X Press.

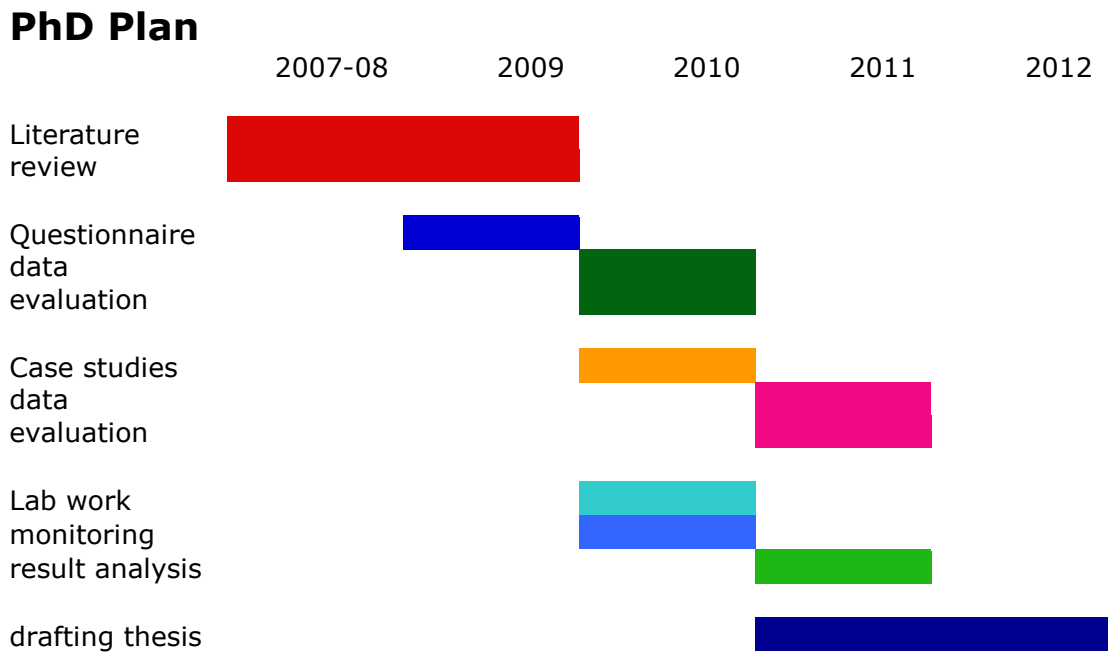
References from the World Wide Web will be cited as follows:-
Cairns Library, 1996 (URL: http://www.medicine.ox.ac.uk/cairns/)

Citing dissertation and theses will follow the basic pattern:-
Last name, first name, year. Title. Degree dissertation, Department name, University name.

Formatting the literature review

The review will be formatted using the 'styles and formatting' menu in Microsoft Word to ensure headings are consistently formatted, font style, font size etc. The contents, figures and tables will be formatted.
Citations will be managed using 'Endnote', or 'Reference Manager'.

Time table



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University of Huddersfield, 2008. *The literature review*.
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University of Edinburgh, 2008. *Undertaking a literature review.*
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National Research Ethics Service, 2008, *Facilitating ethical research.*
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(URL: http://www.eric-on-line.co.uk)

Alejandro R Jadad, professor,[a] Michael Moher, Royal College of General Practitioners research training fellow,[b] George P Browman, professor,[a] Lynda Booker, research assistant,[a] Christopher Sigouin, doctoral student,[a] Mario Fuentes, research assistant,[c] and Robert Stevens, research assistant, 2000.
Systematic reviews and meta-analyses on treatment of asthma: critical evaluation. , British Medical Journal, February 26; 320(7234): 537–540.

Bowling, A., 2002: *Research Methods in Health- investigating health and health services*: Open University Press: ISBN 0-335-20643

Bowling, A., 2005 : *Measuring Health – A review of quality of life measurement scales* : Open University Press: ISBN 0-335-21527

Cairns Library,1996. *A Guide to the Harvard Referencing System*.
(URL: http://www.medicine.ox.ac.uk/cairns/)

Health Protection Agency, 2005. *Providing the Evidence Base for Public Health*. The HPA's Research Strategy 2005-10.

Medical Research Council 2005 : *Good Research Practice* - MRC Ethics series

A1.2 Literature scoring table **(undertaken part way through the research programme)**

Source	Hierachy	Strength
Abramson, M. J., B. G. Toelle, et al., 2013. *Asthma diagnosis and treatment - Is atopy in people aged 40 and over related to fixed airflow obstruction?* World Allergy Organ J 6 Suppl 1: P16(Anto and Sunyer 1995)	3	moderate
AEA Energy and Environment, 2008: *Diffusion Tubes for Ambient NO2 Monitoring : Practical guidance for laboratories and Users*. Report to Defra and the Devolved Administraions ED48673043 Issue 1a.	1	strong
Andersen, Z. J., S. Loft, et al., 2008. *Ambient air pollution triggers wheezing symptoms in infants*. Thorax 63(8): 710-716.	3	moderate
Anees, W., Moore, V.C., Burge, P.S., 2006. *FEV_1 decline in occupational asthma.* Thorax 61:751-755	2	strong
Anto, J., 2004. *The causes of asthma: the need to look at data with different eyes.* Allergy 2004:59 (121-3)	2	strong
http://www.atsjournals.org/doi/full/10.1513/pats.P09ST2#.UoyrA8S-0yo	3	moderate
Ayres, J., Baxter, P., 2004. *Irritant Induced Asthma and RADS*. A short report prepared for Expert Panel on Air Quality Standards.		
http://www.bohrf.org.uk/downloads/Evidence_based_guidance_for_the_assessment_of_new_employees_with_asthma.pdf (2011)	1	strong
Boynton,P., 2004. *Selecting, designing and developing your questionnaire*. BMJ 2004; 328:1312.	5	moderate
http://www.brit-thoracic.org.uk/Portals/0/Guidelines/AsthmaGuidelines/sign101%20Jan%202012.pdf (2011)	1	strong
Brugge, D., J. Vallarino, et al., 2003. *Comparison of multiple environmental factors for asthmatic children in public housing*. Indoor Air **13**(1): 18-27.	2	strong

http://www.bsaci.org/resources/asthma - British Society for Allergy and Clinical Immunology	1	strong
Bylin, G., G. Hedenstierna, et al., 1988. *Ambient nitrogen dioxide concentrations increase bronchial responsiveness in subjects with mild asthma.* Eur Respir J **1**(7): 606-612.	3	moderate
Cameron, V., 2003. *Community Planning in Action – an environmental health perspective*. Environmental Health In Scotland Vol 15 No 4 Winter 2003	7	strong
Cameron, V., 2004. *Corporate Strategy to Community Planning – an exploration of organisational culture within Orkney Islands Council and its impact on the strategic change process*. Napier University, Edinburgh.	3	strong
Cameron, V., Oduyemi, K., 2009. Impacts of Indoor air quality on the health of pre-existing asthmatics at work – a review of litereature and current guidance. Environmental Health In Scotland Vol 21 No 3 Autumn 2009	1	strong
Carlos A. Camargo, Jr., Gary Rachelefsky, and Michael Schatz, 2009. *Managing Asthma Exacerbations in the Emergency Department*, Proceedings of the American Thoracic Society, Vol. 6, No. 4 (2009), pp. 357-366.	3	strong
Carroll, N.G., Mutavdzic, S., James, A.L., 2002. *Increased mast cells and neutrophils in submucosal mucous glands and mucus plugging in patients with asthma.* Thorax 2002:57:677-682	2	strong
Chauhan, A.J., Inskip, H.M., Linklater, C.H. et al, 2003. *Personal exposure to nitrogen dioxide (NO_2) and the severity of virus-induced asthma in children*. The Lancet 361:1939-1944	2	strong
Chinn, S., Davis, D., Burney, P., Luczynska, C., Ackermann-Liebrich, U., Anto, J.M., Cerveri, I., De Marco, R., Gislason, T., Heinrich, J., Janson, C., Kunzli, N., Leynaert, B., Neukirch, F., Schouten, J., Sunyer, J., Svanes, C., Vermeire, P., Wyst, M., 2004. *Increase in diagnosed asthma but not in symptoms in the European Community Respiratory Health Survey*.Thorax 2004:59 :646-651.	1	strong
http://www.colorado.edu/intphys/Class/IPHY3700_Greene/slid	1	moderate

es/generatingContentInterpret/explainPValues.pdf		
COMEAP, 2004. *Guidance on the Effects of Indoor Air Pollutants.* Committee on the Medical Effects of Air Pollution	1	strong
COMEAP, 2006. *Quantification of the Effects of Air Pollutants on Health in the UK*. Committee on the Medical Effects of Air Pollutants.	1	strong
Commission Directive 89/106 EEC, *on the approximation of laws, regulations and administrative provisions of the Member States relating to construction products*.	1	strong
Delfino, R. J., N. Staimer, et al., 2008. *Personal and ambient air pollution exposures and lung function decrements in children with asthma*. Environ Health Perspect **116**(4): 550-558.	2	strong
Department of Health, 2012: *An outcomes Strategy for COPDS and Asthma: NHS Companion Document*	1	strong
Di Giampaolo, L., C. Quecchia, et al., 2011. *Environmental pollution and asthma*. Int J Immunopathol Pharmacol **24**(1 Suppl): 31S-38S.	3	strong
European Community Respiratory Health Survey, 1996.*Variations in the prevalence of respiratory symptoms, self-reported asthma attacks, and use of asthma medication in the European Community Respiratory Health Survey* (ECRHS). European Respiratory Journal; 1996;9:687–95.	3	strong
European Federation of Allergy and Airways Diseases Patients Associations, 2004. *Towards Healthy Air in Dwellings in Europe* (THADE) report. EFA.	1	strong
European Respiratory Society Annual Congress 2103; http://www.ers.org	1	moderate
http://www.erswhitebook.org/	1	strong
European Study of Cohorts for Air Pollution Effects, 2010. ESCAPE: *exposure assessment manual 2010*.Institute for Risk assessment sciences, Utrecht University, Netherlands	1	strong

European Study of Cohorts for Air Pollution Effects, 2008. ESCAPE: *Study manual 2008.* Institute for Risk assessment sciences, Utrecht University, Netherlands	1	strong
Farmer, R., Lawrenson, R., 2004: *Epidemiology in Public Health Medicine*. Blackwell Publishing. ISBN 1-4051-0674-3	1	strong
http://www.gla.ac.uk/sums/users/jdbmcdonald/PrePost_TTest/pandt1.html	1	strong
The Global Asthma Report 2011 http://www.globalasthmanetwork.org/publications/Global_Asthma_Report_2011.pdf	1	strong
Hasselblad, V., Eddy, D.M., Kotchmar, D.J., 1992. *Synthesis of environmental evidence: nitrogen dioxide epidemiology studies*. Journal of the Air Waste Management Association 1992:42:662-671	2	strong
Health and Safety Commission, 2005. *A Strategy for Workplace Health and Safety in Great Britain to 2010 and beyond.*	1	strong
Health and Safety Executive, 2003. *Occupational Asthma Research Issues*. Report from a HSE workshop January 2003 – HSL/2003/05	1	strong
Health and Safety Executive, 2006. *The true cost of occupational asthma in Great Britain*. Research report 474.	1	strong
Health and Safety Executive, 2008: *Irritancy and sensitisation*. Research report 601	1	strong
Health and Safety Executive/Local Authorities (HELA*) Advice to Local Authorities on Intervention Programmes and an Inspection Rating System*. Circular Number LAC 67/1 (rev).	1	strong
Hetes, R.G., Pierson, T.K.,1991. *A framework for Risk Characterisation of Environmental Pollutants.* Research Triangle Institute, North Carolina, United States of America.	1	strong

House of Commons Environmental Audit Committee, 2009-10. *Air Quality*. Fifth report of session 2009-10.	1	strong
House of Lords Science and Technology Committee, 2007. *'Allergy'* - Volume 1 Report.	1	strong
NHS Health Scotland and Health Protection Scotland, 2005. *Internal Air Quality and Health.*	1	strong
International Study of Asthma and Allergies in Childhood (ISAAC) Steering Committee. *Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema*: ISAAC. Lancet 1998;351:1225–32.	1	strong
Lin, M., Y. Chen, et al., 2004. *Gaseous air pollutants and asthma hospitalization of children with low household income in Vancouver, British Columbia, Canada*. Am J Epidemiol **159**(3): 294-303. .	3	strong
Linaker, C. H., D. Coggon, et al., 2000. *Personal exposure to nitrogen dioxide and risk of airflow obstruction in asthmatic children with upper respiratory infection*. Thorax **55**(11): 930-933	3	strong
Lioy, P.J.,1990. *Assessing total human exposure to contaminants*. Environ Sci Technol 24(7):938-94		
Matthews,I., Parry, S., 2005 : *The burden of disease attributable to environmental Pollution.* University of Wales College of Medicine	1	strong
http://www.medicinenet.com/allergy/page4.htm#asthma	1	strong
Molhave, L., 1991. *Volatile organic compounds, indoor air quality and health*. Indoor air 1991-4 357-76	2	strong
http://guidance.nice.org.uk/QS25	1	strong
NHS Health Scotland, 2005. *Health in Scotland 2005* Scottish Executive	1	strong
http://www.nhs.uk/Conditions/Allergies/Pages/Causes.aspx	1	strong

http://www.hse.gov.uk/asthma/	1	strong
Nishimura, K. K., J. M. Galanter, et al., 2013. *Early Life Air Pollution and Asthma Risk in Minority Children: The GALA II & SAGE II Studies*. Am J Respir Crit Care Med	3	strong
Neville, R.G., Hoskins, G., Smith, B., McCowan, C., 2003. *The economic and human costs of asthma in Scotland*. The Primary Care Respiratory Journal, 12(4): 115-118	1	strong
The Parliamentary Office of Science and Technology, 2000. *Indoor allergens and asthma*. Post note 152, December 2000	1	strong
Prescott,G.J., Lee, R.J., Cohen,G.R., Elton, R.A.,A J Lee, A.J., Fowkes, F.G.R., Agius, R.M., 2000. *Investigation of factors which might indicate susceptibility to particulate air pollution.* BMJ Publishing Group Ltd. *Occup Environ Med* 2000, **57:**53-57 (January)	3	strong
Price,G., 2007, *Effects of weather, air quality, and geographical location on asthma and COPD exacerbations in the localities of Worcester and Dudley.* Coventry University.	2	strong
Primary Care Commissioning, 2012*: Designing and Commissioning Services for Adults with Asthma: A Good Practice Guide*. http://www.pcc-cic.org.uk/article/designing-and-commissioning-services-adults-asthma-good-practice-guide	1	strong
Royal College of Physicians, 2003*: Allergy the unmet need.* RCP 2003. http://www.rcplondon.ac.uk/sites/default/files/documents/allergy-unmet-need-2003.pdf	1	strong
Royal Environmental Health Institute of Scotland, 2007. *Environmental Health in Scotland – a Workforce in Crisis – the way forward*. 2007	1	moderate

Rusznak,C.,Devalia,J.L., Davies,R.J., 1996, *Airway response of asthmatic subjects to inhaled allergen after exposure to pollutants*: Thorax 1996;51:1105-1108	3	strong
Samet, J. M. and M. L. Bell, 2004*. Commentary: nitrogen dioxide and asthma redux.* Int J Epidemiol **33**(1): 215-216	3	strong
SRUC, 2013. *Research hypotheses* – Scotland's Rural University College - student training module.	1	strong
Sibbald, B, Anderson, H.R., McGuigan, S., 1992. *Asthma and employment in young adults*. Thorax 1992;47:19-24	3	strong
States of Guernsey, 2001. *Guernsey Census – report on the census of the population and households.*	1	strong
States of Guernsey, 2010. *Air Quality in Guernsey – Update Screening and Assessment 2010.* Office of Environmental Health and Pollution Regulation	1	strong
https://students.shu.ac.uk/lits/it/documents/pdf/questionnaire_analysis_using_spss.pdf	1	moderate
Tunnicliffe, W.S., Burge, P.S., Ayres, J.G., 1994. *Effect of domestic concentrations of nitrogen dioxide on airway responses to inhaled allergen in asthmatic patients*. The Lancet 24-31: 344: 1733-6	2	strong
http://uk-air.defra.gov.uk/reports/cat06/0502160851_Conversion_Factors_Between_ppb_and.pdf	1	strong
US Expert Panel Report 3, 2007. *Guidelines for the Diagnosis and Management of Asthma*. Bethesda (MD): National Institutes of Health (US), National Heart, Lung, and Blood Institute (NHLBI)	1	strong
US Environment Protection Agency 2013 http://www.epa.gov/iaq/no2.html	1	strong
US Environment Protection Agency, 2012. Exposure Model for	1	moderate

Individuals (EMI)		
http://www.webmd.com/asthma/guide/what-is-asthma	1	strong
Weisel, C.P., 2002. *Assessing exposure to air toxics relative to asthma*. Environ. Health Perspect. 2002;110, Suppl 4.	2	strong
World Health Organization, 2002. *Quantification of the Health Effects of Exposure to Air Pollution*	1	strong
World Health Organisation, 2002. *Role of Human Exposure Assessment in Air Quality Management.* Report of the Joint Working Group WHO Joint Research Centre European Concerted Action Urban Air, Indoor Environment and Human Exposure.	1	strong
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World Health Organisation, 2005. *WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulphur dioxide*. Global update 2005	1	strong
World Health Organisation, 2006. *Preventing Disease through Healthy Environments- Towards an estimate of the environmental burden of disease*.	1	strong
World Health Organisation 2006. *Development of WHO Guidelines for Indoor Air Quality*. WHO Regional Office for Europe	1	strong

A1.3 UK Air Quality Objectives 2007

Summary of objectives of the UK Air Quality Strategy 2007			
Pollutant	**Objective**	**Measured as**	**To be achieved by**
Benzene All Authorities	16.25 µg/m^3	Running Annual Mean	31 December 2003
Benzene Authorities in England and Wales only	5 µg/m^3	Annual Mean	31 December 2010
Benzene Authorities in Scotland and Northern Ireland only	3.25 µg/m^3	Running Annual Mean	31 December 2010
1,3-Butadiene	2.25 µg/m^3	Running Annual Mean	31 December 2003
Carbon monoxide Authorities in England, Wales and Northern Ireland only	10.0 mg/m^3	Maximum daily running 8 Hour Mean	31 December 2003
Carbon monoxide Authorities in Scotland only	10.0 mg/m^3	Running 8 Hour Mean	31 December 2003
Lead	0.5 µg/m^3	Annual Mean	31 December 2004
	0.25 µg/m^3	Annual Mean	31 December 2008
Nitrogen dioxide	200 µg/m^3 Not to be exceeded more than 18 times per year	1 Hour Mean	31 December 2005
	40 µg/m^3	Annual Mean	31 December 2005
Nitrogen Oxides**	(V) 30 µg/m^3	Annual Mean	31 December 2000
Ozone*	100 µg/m^3	Running 8 hour Mean Daily maximum of running 8 hr mean not to be exceeded more than 10 times per year	31 December 2005
Particles (PM_{10}) (gravimetric) All authorities	50 µg/m^3 Not to be exceeded more than 35 times per year	24 Hour Mean	31 December 2004
	40 µg/m^3	Annual Mean	31 December 2004
Particles (PM_{10}) Authorities in Scotland only	50 µg/m^3 Not to be exceeded more than 7 times per year	24 Hour Mean	31 December 2010

	18 µg/m^3	Annual Mean	31 December 2010
Particles ($PM_{2.5}$) Exposure Reduction Authorities in UK (except Scotland)	25 µg/m^3	Annual Mean	2020
Particles ($PM_{2.5}$) Exposure Reduction Authorities in Scotland only	12 µg/m^3	Annual Mean	2020
Particles ($PM_{2.5}$) Exposure Reduction Authorities in UK urban areas	Target of 15% reduction in concentrations at urban background	Annual Mean	Between 2010 and 2020
Polycyclic aromatic hydrocarbons All authorities	0.25 ng/m^3	Annual Mean	31 December 2010
Sulphur dioxide	266 µg/m^3 Not to be exceeded more than 35 times per year	15 Minute Mean	31 December 2005
	350 µg/m^3 Not to be exceeded more than 24 times per year	1 Hour Mean	31 December 2004
	125 µg/m^3 Not to be exceeded more than 3 times per year	24 Hour Mean	31 December 2004
	(V) 20 µg/m^3	Annual Mean	31 December 2000
	(V) 20 µg/m^3	Winter Mean (01 October – 31 March)	31 December 2000

µg/m^3 – micrograms per cubic metre mg/m^3 – milligrams per cubic metre

*Ozone is not included in the Regulations ** Assuming NO_x is taken as NO_2

A1.4 Air Quality Standards Regulations 2010- Limit Values

Pollutant	Averaging Period	Limit Value	Margin of Tolerance
Sulphur Dioxide	One hour	350 µg/m^3 not to be exceeded more than 24 times a calendar year	150 µg/m^3 (43%)
	One day	150 µg/m^3 not to be exceeded more than 3 times a calendar year	
Nitrogen Dioxide	One hour	200 µg/m^3 not to be exceeded more than 18 times a calendar year	
	Calendar year	40 µg/m^3	
Benzene	Calendar year	5 µg/m^3	
Carbon Monoxide	Maximum daily 8 hour mean	10 mg/m^3	
Lead	Calendar year	0.5 µg/m^3	100%
Particles (PM_{10})	One day	50 µg/m^3 not to be exceeded more than 35 times a calendar year	50%
	Calendar year	40 µg/m^3	20%
Particles ($PM_{2.5}$)	Calendar year	25 µg/m^3 to be achieved by 1 January 2015	20% on 11th June 2008, decreasing on the next 1st January and every 12 months thereafter by equal annual percentages to reach 0% by 1st January 2015

A1.5 Target Values

Pollutant	Target Value for the Total Content in the PM_{10} Fraction Averaged Over a Calendar Year	Date by Which Target Value Should Be Met
Arsenic	6 ng/m^3	31 December 2012
Cadmium	5ng/m^3	31 December 2012
Nickel	20 ng/m^3	31 December 2012
Benzo(a)pyrene	1 ng/m^3	31 December 2012

Pollutant	Objective	Averaging Period	Target Value
Ozone	Protection of human health	Maximum daily eight hour mean	120 µg/m^3 not to be exceeded on more than 25 days per calendar year averaged over three

			years
	Protection of vegetation	May to July	AOT 40 (calculated from 1 h values) 18000 µg/m^3 .h averaged over five years

Pollutant	Averaging Period	Target Value
Particles ($PM_{2.5}$)	Calendar Year	25 µg/m^3

Appendix 2

A2 .1 Ethics approval from Abertay University

From: "Gray, James" <J.Gray@abertay.ac.uk>
To: "CAMERON, VALERIE" <0606122@abertay.ac.uk>
Cc: "Oduyemi, Kehinde" <K.Oduyemi@abertay.ac.uk>
Sent: 08 June 2009 10:55
Subject: E1 Ethical Considerations Application

Your E1 Ethical Considerations application has been given unconditional approval and your research may now commence.
A copy of your E1 form has been lodged with your supervisor, Dr Oduyemi.
Thank you.

James Gray
Administrative Assistant
Contemporary Sciences
University of Abertay Dundee

Appendix 3

A3.1 **NO_2 monitoring data - Diffusion tube monthly mean – 2008-2012**

A3.2 **Real time NOX data 2010-2012 - CD Rom attachment**

A3.3 **Real-time NO_2 data 2010 – Lukis House, The Grange, shown as hourly mean in ppb (monthly graphs).**

A3.4 **Hospital admissions data**

A3.5 **Hospital admissions per parish, standardised rates for 2008-2012**

A3.6 **Descriptive statistics for calculations**

A3.1 NO_2 Monitoring Data - diffusion tube monthly mean 2008-2012

2008

			Period Ending	30/01/2008	27/02/2008	02/04/2008
SPP 1	Roadside Urban	College Street (St. James)		6.72	17.20	16.92
SPP 2	Roadside Urban	Fountain Street		27.13	26.32	26.18
SPP 3	Background Urban	Commercial Arcade		6.87	9.86	8.49
SPP 4	Roadside Urban	Albert Statue			15.51	17.26
SPP 5	Roadside Urban	Trinity Square		11.06	13.14	11.59
STS 1	Roadside Urban	South Side		11.30	14.18	12.21
STS 2	Background Rural	Les Quatre Vents, La Passee		3.51	6.16	5.39
FOR 1	Background Rural	Near Corbiere		1.37	4.62	2.67
STM 2	Background Urban	Princess Elizabeth Hospital		2.67	6.57	

30/04/2008	28/05/2008	02/07/2008	30/07/2008	03/09/2008	01/10/2008	29/10/2008	03/12/2008	07/01/2009
16.92	26.82	14.63	13.22	9.18	20.96	11.24	15.15	17.25
26.18	37.19	24.91	17.69	18.51	19.30	20.96	16.14	22.41
8.49	10.99	7.55	6.35	4.97	7.86	6.87	8.80	10.10
17.26	18.44	16.87	15.13	12.62	11.65	15.66	14.55	15.16
11.59	16.64	10.90	8.52	8.75	10.66	11.04	11.06	13.12
12.21	14.33	11.17	9.31	9.24	8.55	10.09	12.29	11.34
5.39	6.38	4.64	3.34	2.35	3.10	2.24	4.13	5.49
2.67	4.00	2.78	2.52	1.46	3.05	1.54	2.62	4.80
4.94	9.16	4.76	3.89	2.46	4.78	3.41	5.00	6.57

2009		Period Ending	04/02/2009	04/03/2009
SPP 1	Roadside Urban	College Street (St. James)	13.89	14.96
SPP 2	Roadside Urban	Fountain Street	24.09	24.01
SPP 3	Background Urban	Commercial Arcade	8.53	10.55
SPP 4	Roadside Urban	Albert Statue	13.04	15.41
SPP 5	Roadside Urban	Trinity Square	13.46	13.79
STS 1	Roadside Urban	South Side	12.75	12.21
STS 2	Background Rural	Les Quatre Vents, La Passee	5.57	5.18
FOR 1	Background Rural	Near Corbiere	4.95	4.19
STM 2	Background Urban	Princess Elizabeth Hospital	5.66	6.53

01/04/2009	29/04/2009	03/06/2009	01/07/2009	29/07/2009	02/09/2009	30/09/2009	04/11/2009	02/12/2009	06/01/2010
11.43	17.49	16.80	21.78	8.58	10.92	18.60	14.74	7.98	
31.43	32.26	26.76	36.46	23.46		27.71	24.79	16.80	22.34
	9.05	7.84	9.16	4.61	4.94	7.93	8.01	4.93	8.16
15.42	15.34	14.04	15.33	14.31	14.25	14.03	13.10	10.98	11.86
16.21	14.07	11.97	14.10	7.50	9.25	12.91	11.78	8.27	10.07
14.67	13.97	8.94	11.16	8.96	8.88	9.10	11.46	7.56	9.85
6.40	5.78	3.87	4.93	2.58	2.53	3.46	4.27	2.29	3.92
3.89	3.45	2.42	3.11		1.72	3.16	3.09	1.33	2.31
6.48		7.45	6.03	2.51	2.93	6.18	4.91	2.52	4.44

2010		Period Ending	03/02/2010	03/03/2010	31/03/2010
SPP 1	Roadside Urban	College Street (St. James)	20.57	21.26	17.77
SPP 2	Roadside Urban	Fountain Street	38.44	32	22.69
SPP 3	Background Urban	Commercial Arcade	11.09	11.41	11.42
SPP 4	Roadside Urban	Albert Statue	24.87	15.37	16.98
SPP 5	Roadside Urban	Trinity Square	14.24	14.89	11.11
STS 1	Roadside Urban	South Side	14.87	15.21	13.67
STS 2	Background Rural	Les Quatre Vents, La Passee	5.85	6.53	6.8
STS 3	Roadside Urban	Les Banques, Red Lion			
VAL 1	Roadside Urban	Vale Avenue			
FOR 1	Background Rural	Near Corbiere	2.85	4.16	3.77
STM 2	Background Urban	Princess Elizabeth Hospital	7.13	8.46	7.4

28/04/2010	02/06/2010	30/06/2010	04/08/2010	01/09/2010	29/09/2010	03/11/2010	01/12/2010	05/01/2011
27.89	13.85	25.7	11.17	7.46	18.7		22.2	23.4
38.77	22.92	38.08	26.53			31.6	27.2	
12.39	14.17	12.06	5.2	4.92	6.8	9.3	10.3	11.6
15.78	31.44	13.8	10.54	17.61	15.4	18.3	17.4	16.3
15.46	17.64	16.34	9.65	10.47	12.3	13.1	15.2	15.5
16.52	28.09	10.63	7.36	9.2	11	11.8	14.9	15.5
	6.26	6.51	1.9	3.33	3.2	4.4	5.7	6.3
		22.21	12.31	5.87	8.6	15.7	19.3	20.8
		19.36	23.74			26.5	27.7	6.3
		8.71	3.93		2.1		3.1	4.3
	7.59	4.44	3.05	4.28	4.3	5.1	7.1	8.2

2011		Period Ending	02/02/2011	02/03/2011	30/03/2011
SPP 1	Roadside Urban	College Street (St. James)	20.90	14.90	29.90
SPP 2	Roadside Urban	Fountain Street	31.20	30.20	37.70
SPP 3	Background Urban	Commercial Arcade	9.90	8.20	12.30
SPP 4	Roadside Urban	Albert Statue	16.90	16.80	2.90
SPP 5	Roadside Urban	Trinity Square	15.20	12.30	15.60
STS 1	Roadside Urban	South Side	14.20	13.00	18.50
STS 2	Background Rural	Les Quatre Vents, La Passee	6.00	4.90	8.20
STS 3	Roadside Urban	Les Banques, Red Lion	26.10	21.90	23.60
VAL 1	Roadside Urban	Vale Avenue	28.10	26.30	29.10
FOR 1	Background Rural	Near Corbiere	3.60	3.00	5.70
STM 2	Background Urban	Princess Elizabeth Hospital	8.40	4.90	9.60

27/04/2011	01/06/2011	29/06/2011	03/08/2011	31/08/2011	28/09/2011	02/11/2011	30/11/2011	04/01/2012
24.10	13.70	12.18	17.05	7.20	16.61	14.01	15.55	9.20
33.10	28.10	16.64		22.20	25.78		31.17	23.16
11.60	6.50	3.57	5.43	5.50	6.75	7.87	9.24	5.00
16.10	14.80	9.31	14.66	10.90	17.38		16.08	14.45
14.40	9.60	8.95	10.31	14.70	10.88	12.37	13.00	8.61
16.40	10.00	10.65	8.05	7.50	10.86	13.16	12.94	8.47
6.90	3.00	3.45	3.87	2.60	3.37	4.41	6.10	2.10
19.40	10.10	9.50	12.84	8.10	12.55	17.24	21.45	8.12
25.00	22.10	21.38	17.14	25.70	25.97	23.80	23.92	20.06
3.70	2.30	2.60	3.30	1.70	2.29	2.71	4.01	1.57
4.10	3.90	2.78	4.52	2.90	4.29	4.48	6.74	2.25

2012		Period Ending	01/02/2012	29/02/2012	28/03/2012
SPP 1	Roadside Urban	College Street (St. James)	18.29	22.00	26.00
SPP 2	Roadside Urban	Fountain Street	26.25	28.40	31.00
SPP 3	Background Urban	Commercial Arcade	17.92	9.10	10.50
SPP 4	Roadside Urban	Albert Statue	17.17	17.90	19.10
SPP 5	Roadside Urban	Trinity Square	10.51	15.50	17.70
STS 1	Roadside Urban	South Side	12.46	13.70	15.50
STS 2	Background Rural	Les Quatre Vents, La Passee	5.67	6.10	7.30
STS 3	Roadside Urban	Les Banques, Red Lion	13.73	18.40	21.40
VAL 1	Roadside Urban	Vale Avenue	22.29		28.00
FOR 1	Background Rural	Near Corbiere	4.18	4.40	4.90
STM 2	Background Urban	Princess Elizabeth Hospital	5.11	7.00	7.80

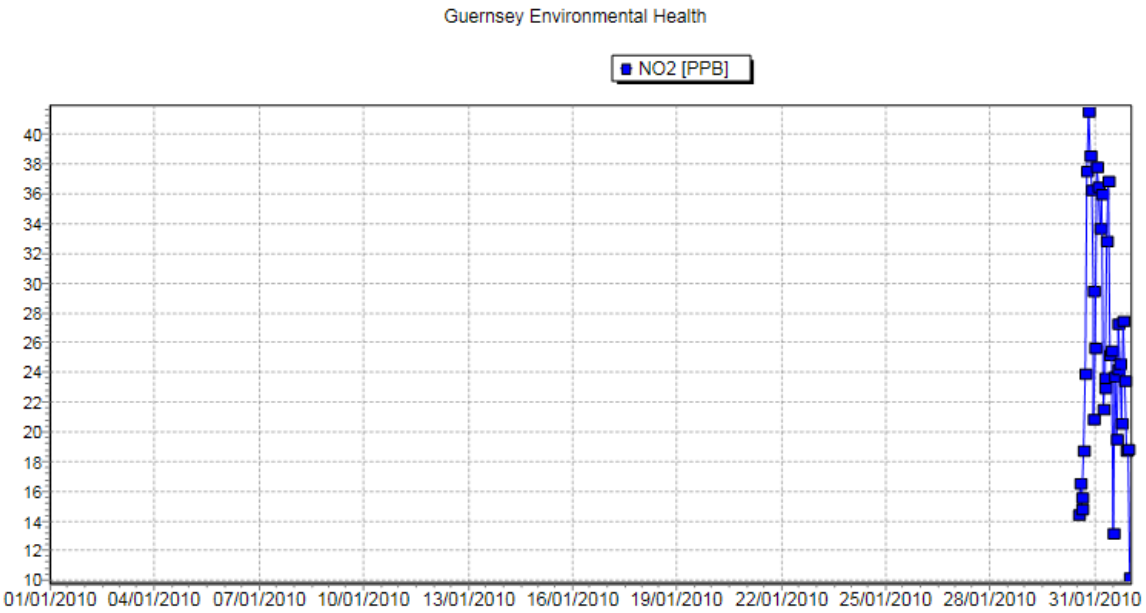
25/04/2012	30/05/2012	27/06/2012	01/08/2012	29/08/2012	26/09/2012	31/10/2012	28/11/2012	02/01/2013
20.00	23.20	20.80	16.40	18.90	21.70		18.40	16.00
29.40	30.80	28.50	24.00		26.90	17.60	18.60	
8.00	8.40	6.30	5.80	6.50	7.40	8.70	8.40	6.10
17.40	16.10	15.40	13.80	16.50	23.00	19.70	24.50	18.80
12.90	12.50	11.70	10.60	11.40	14.30	14.80	12.60	10.50
12.50	13.60	10.90	9.70	10.30	12.00	12.80	16.00	12.10
4.30	5.10	5.50	4.40	4.30	4.90	5.20	5.70	3.70
15.30	23.70	20.80	15.90	17.30	15.00	16.70	17.80	14.60
	22.80		20.60	28.60	26.60	26.70	27.70	23.20
4.30	4.50	3.90	4.30	2.80	4.10		3.20	2.90
5.60	7.50	5.30	6.10	5.40	5.70	6.60	5.30	4.40

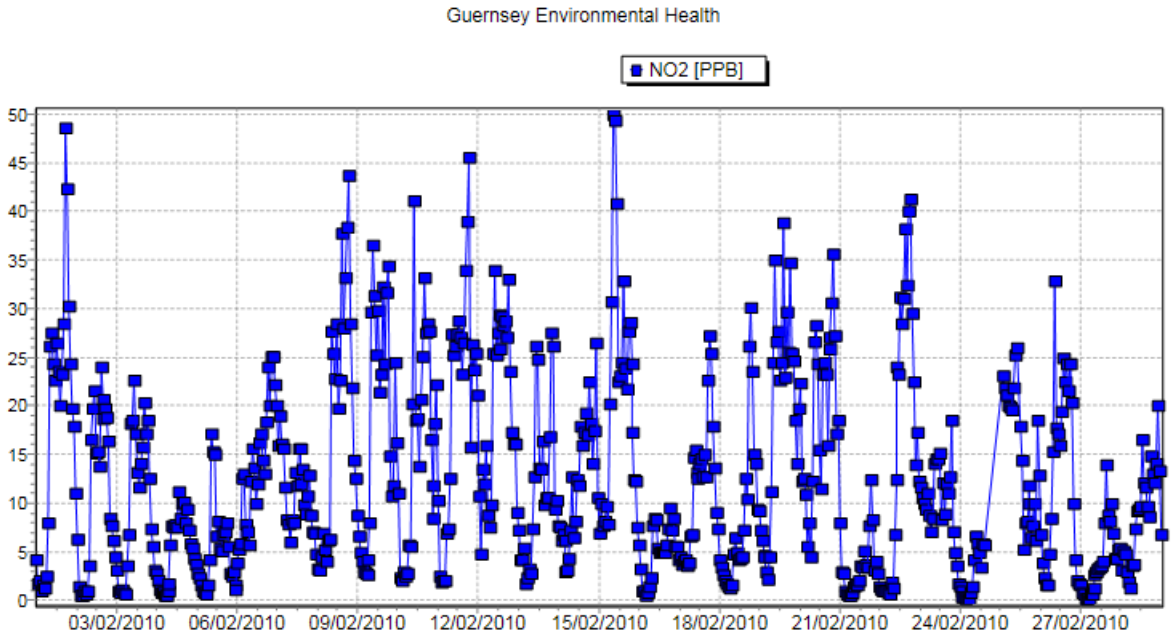
A3 .2 Real time NOX data 2010-2012 - CD Rom attachment

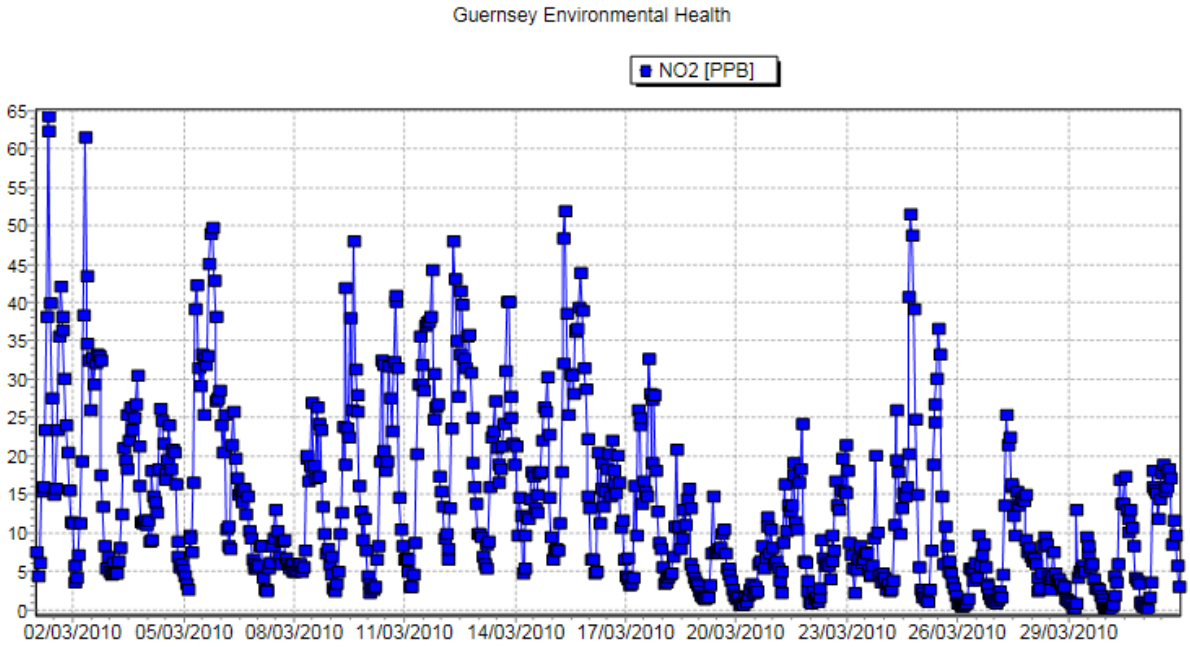
Further copies are available from

vcameron@hssd.gov.gg

A3.3 Real-time NO_2 data 2010 – Lukis House, The Grange, shown as hourly mean in ppb.



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17/03/2010
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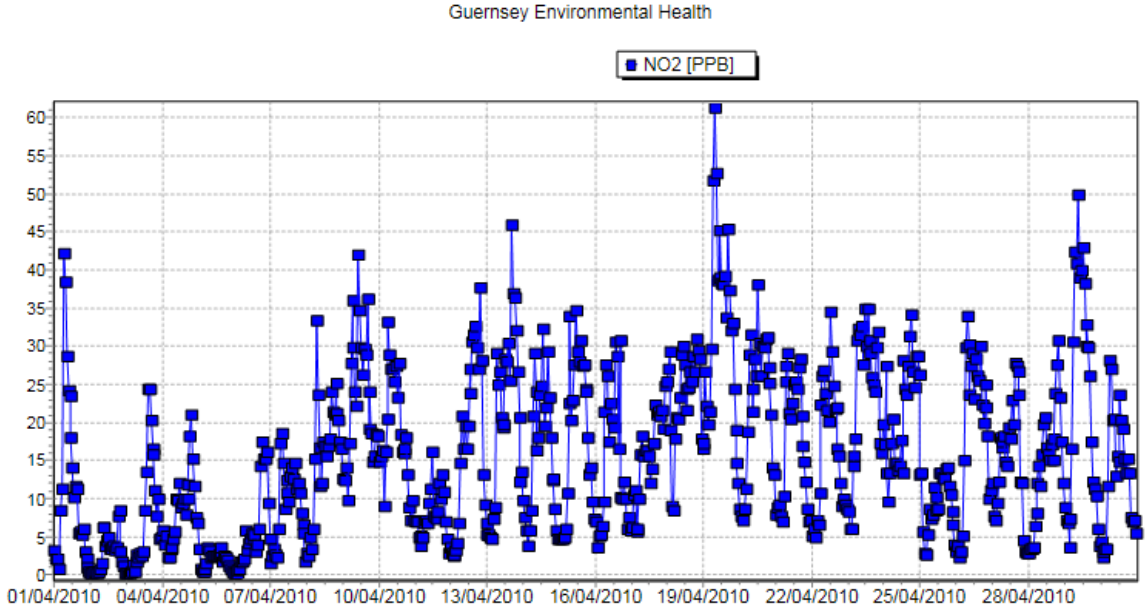
23/03/2010
26/03/2010
29/03/2010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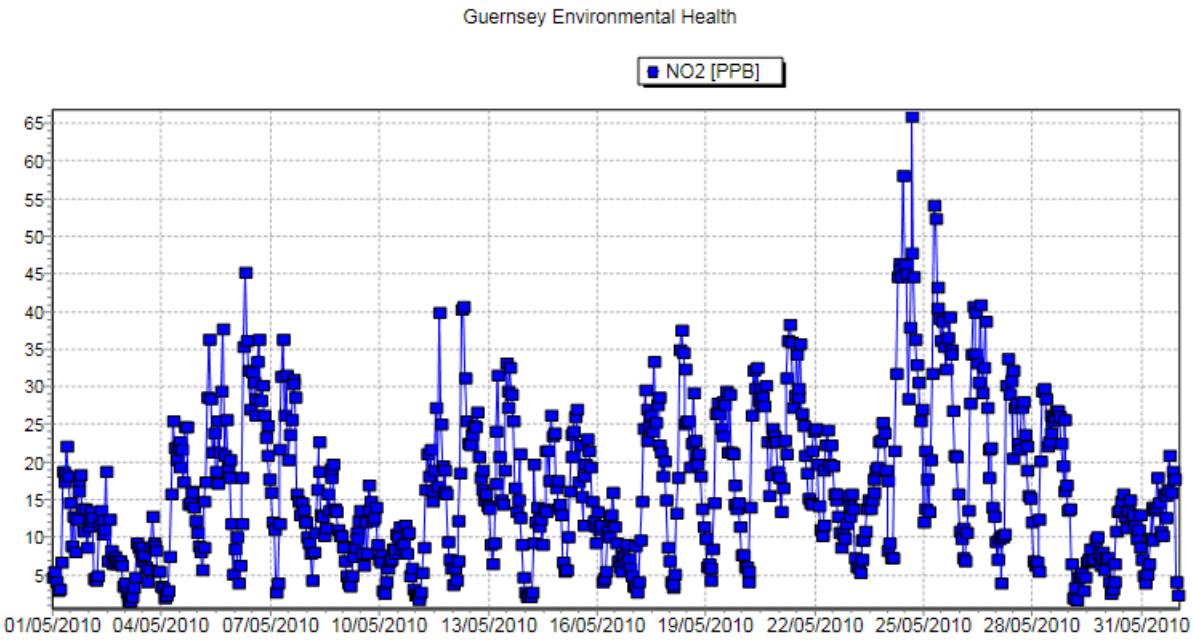
16/04/2010
19/04/2010
22/04/2010
25/04/2010
28/04/2010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28/05/2010
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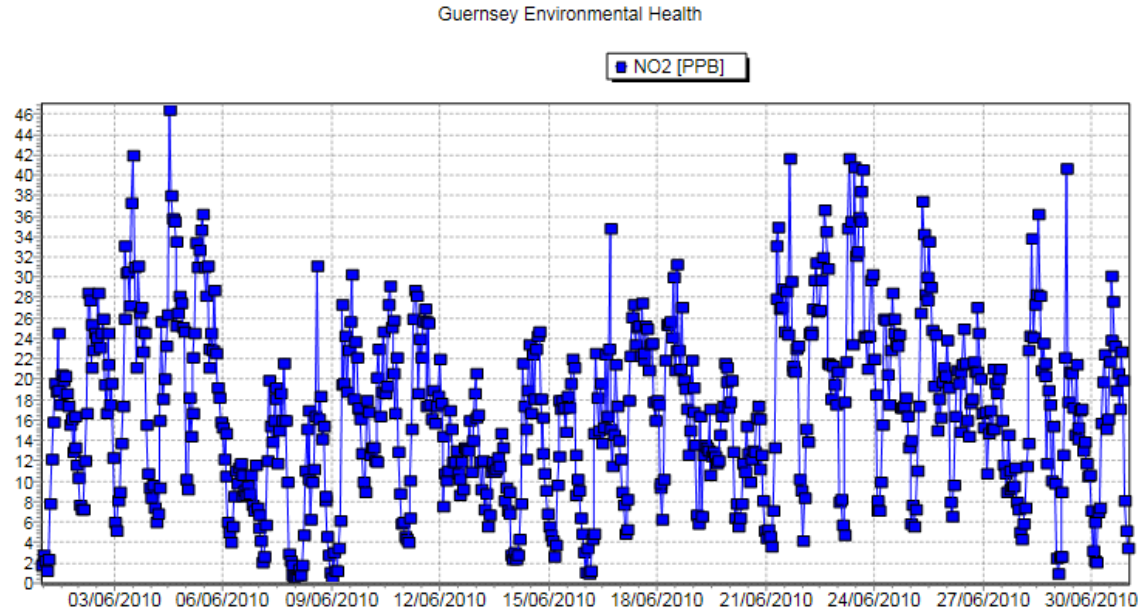
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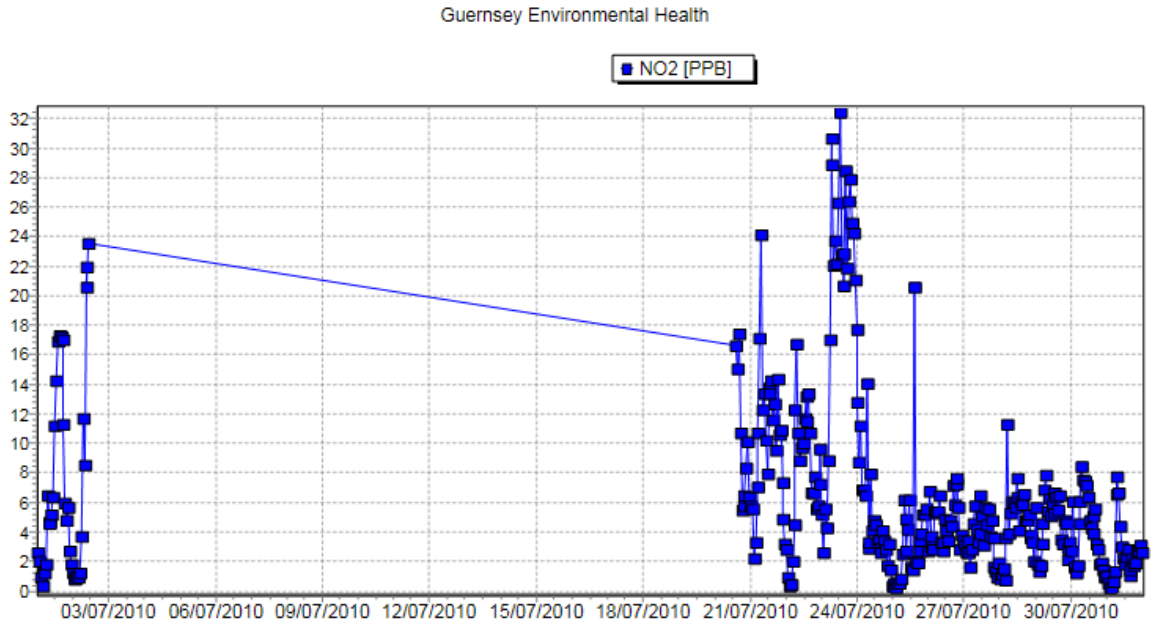
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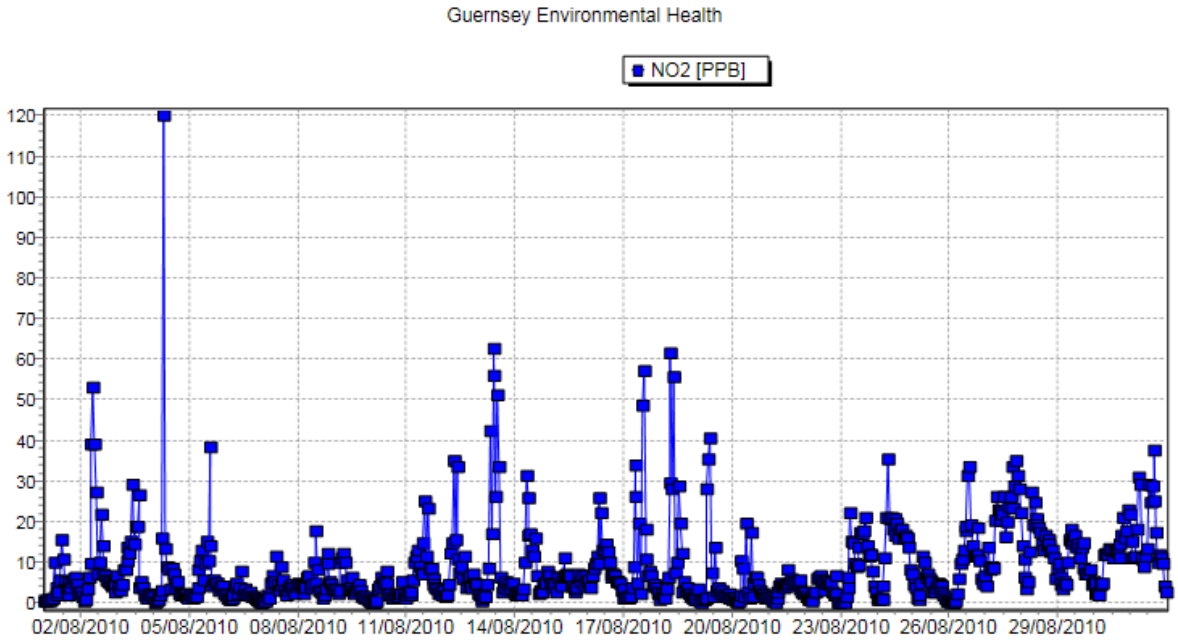
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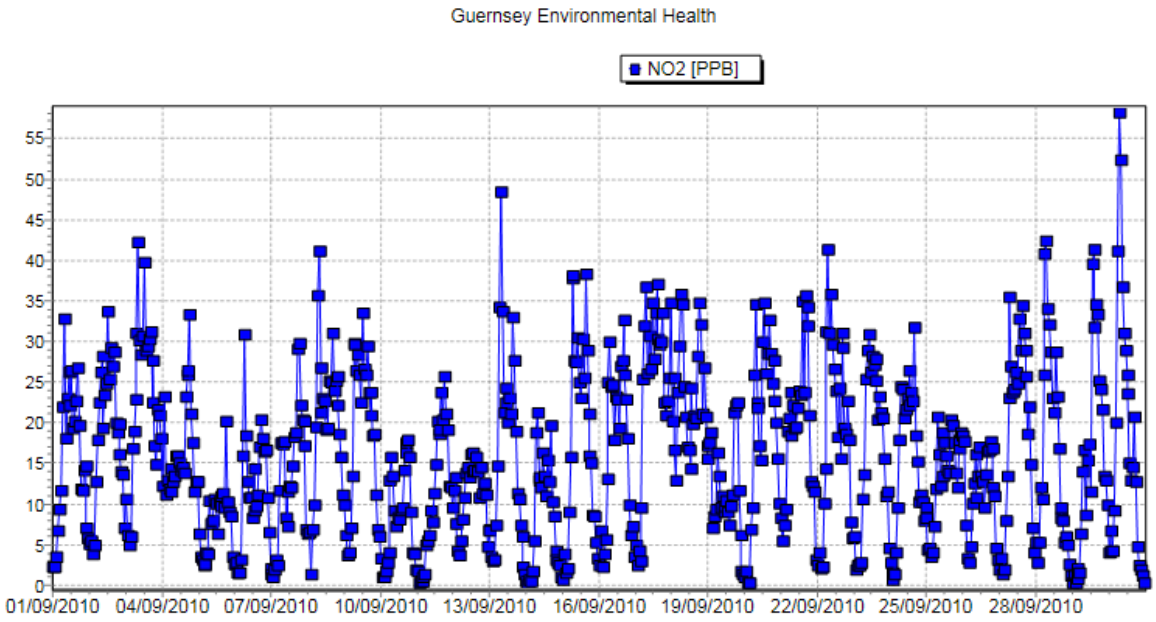
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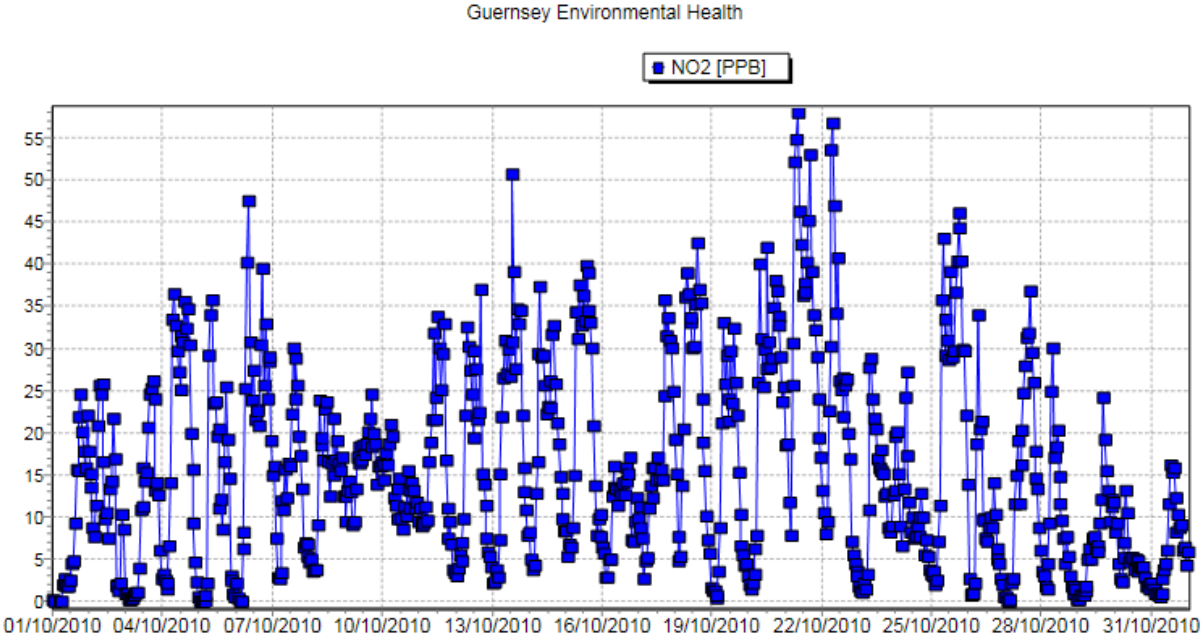
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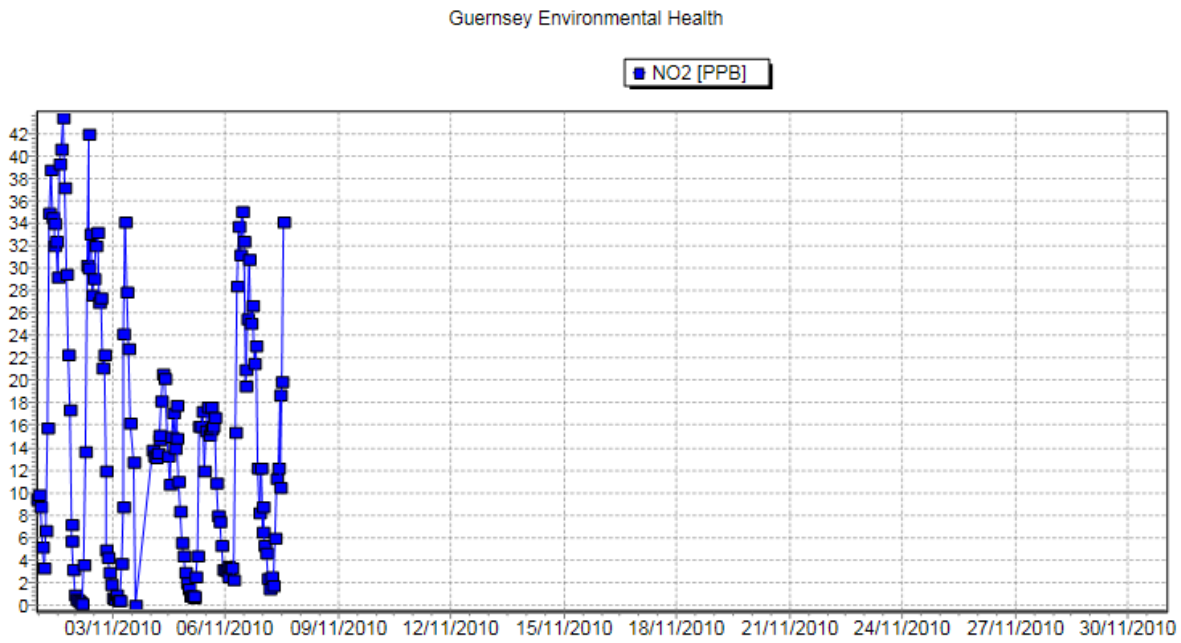
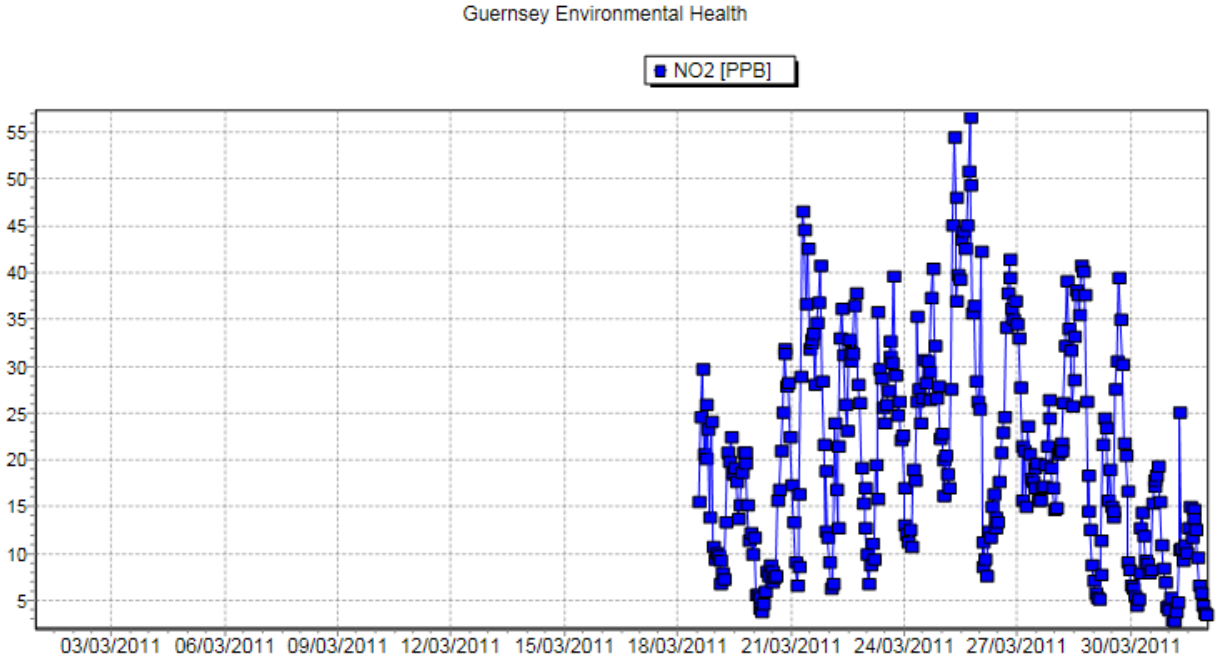
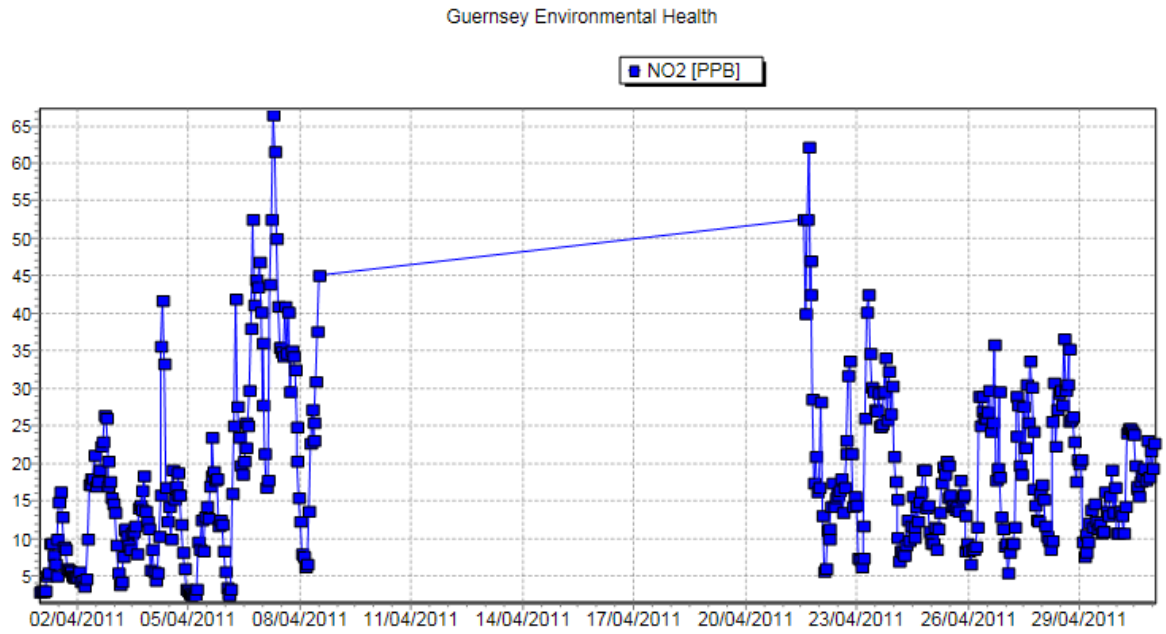


NO_2 Data for 2011 – Lukis House, the Grange

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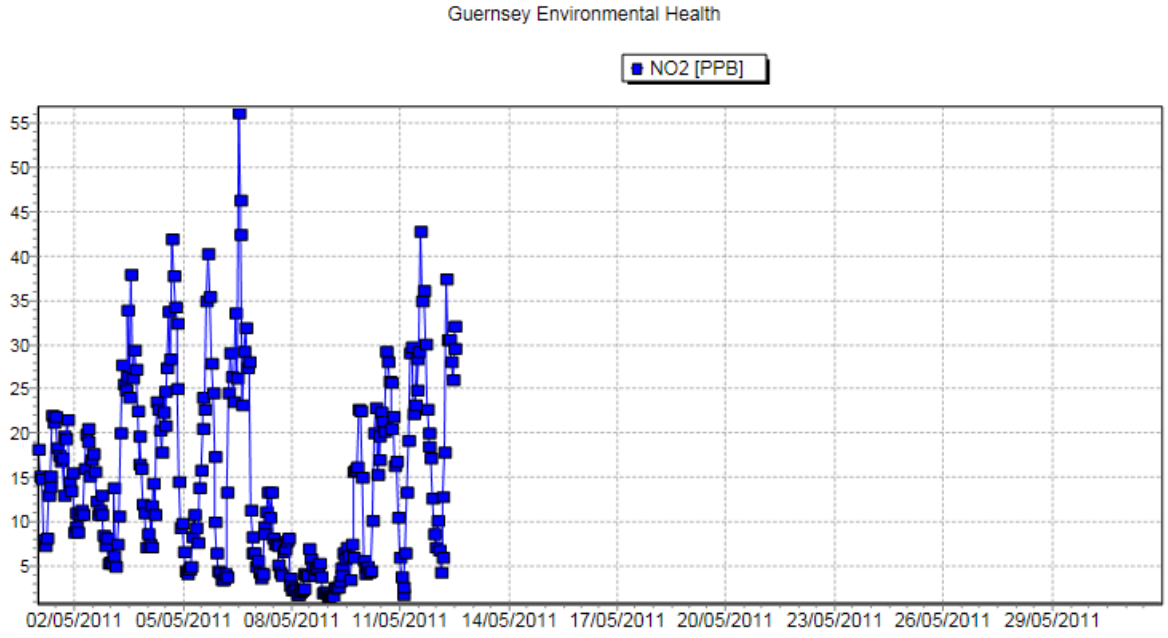
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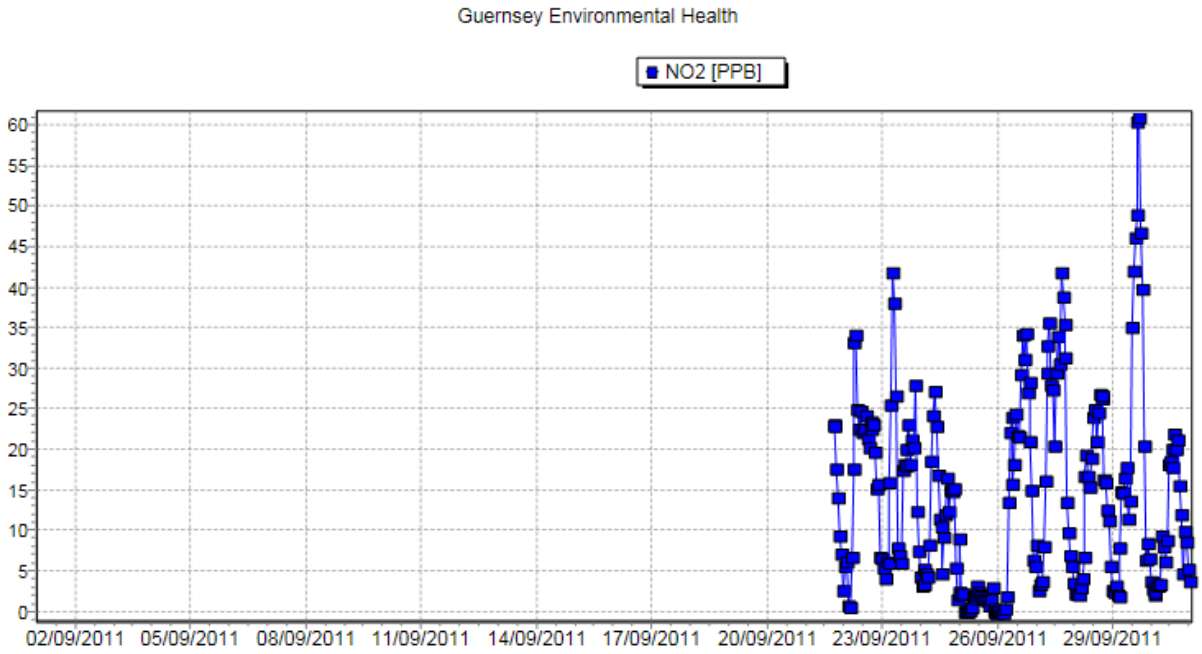
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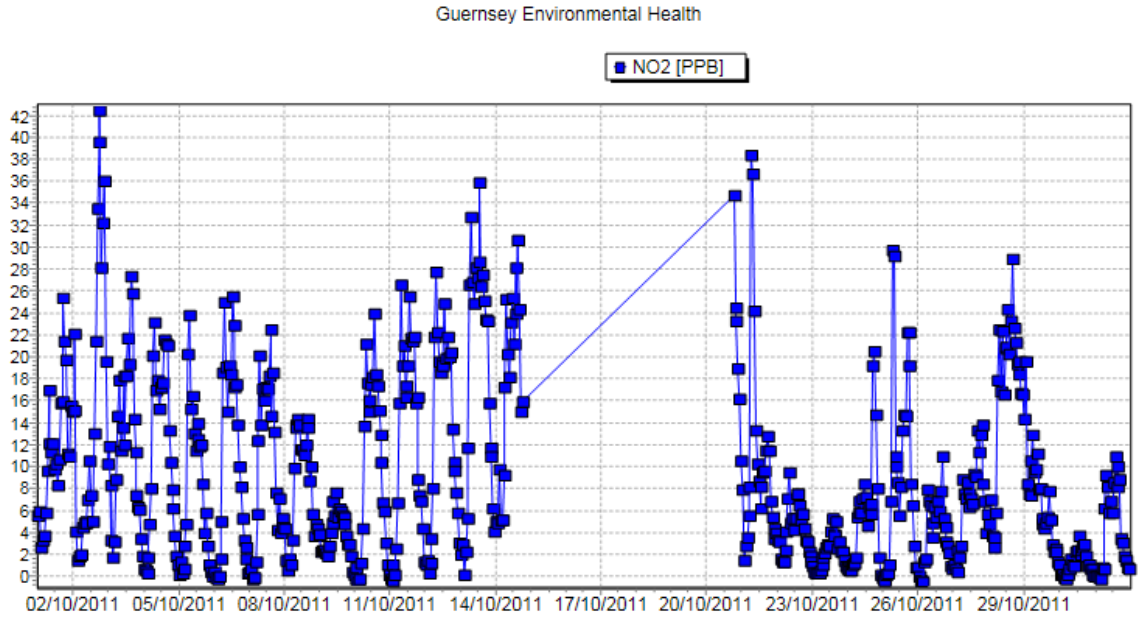
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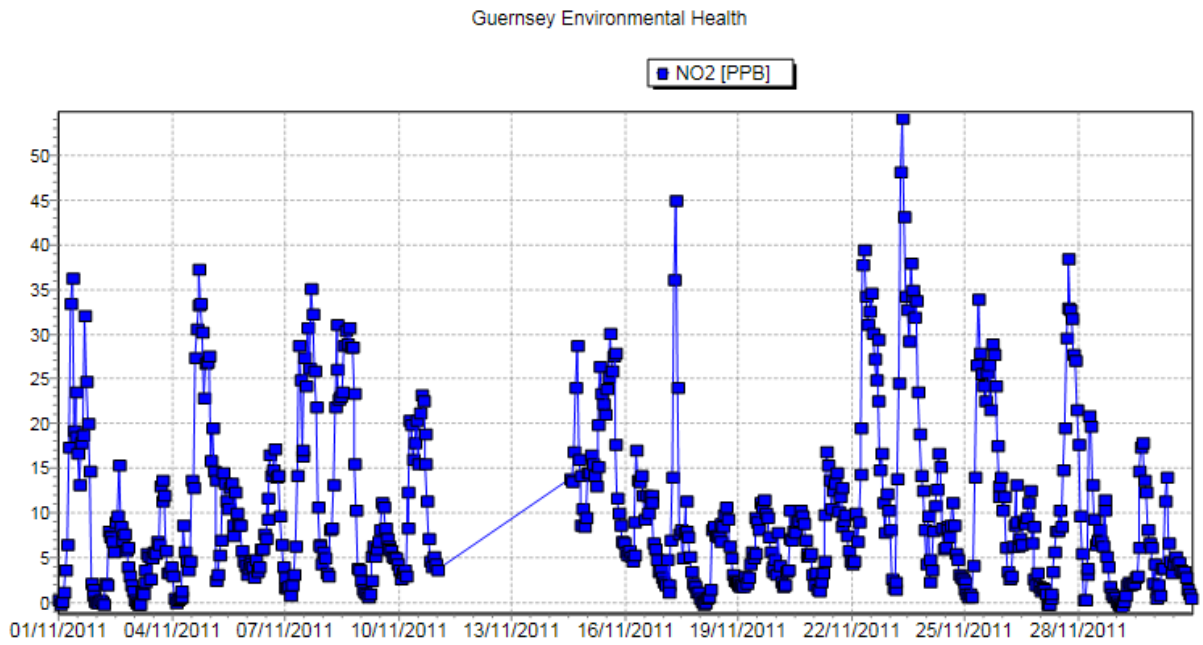
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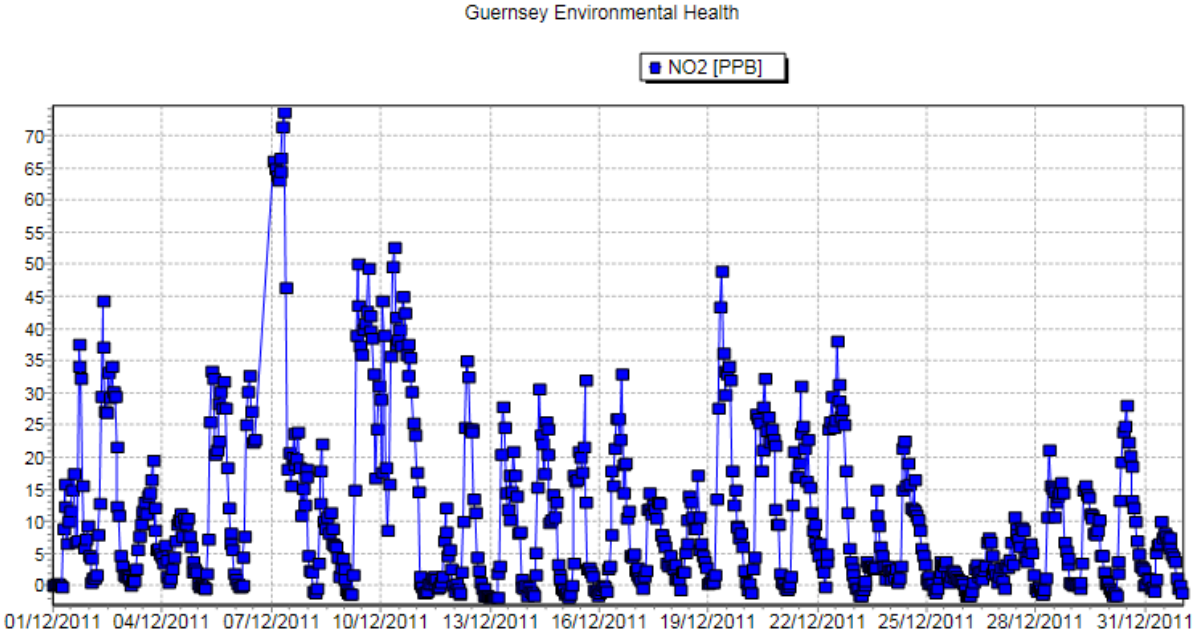
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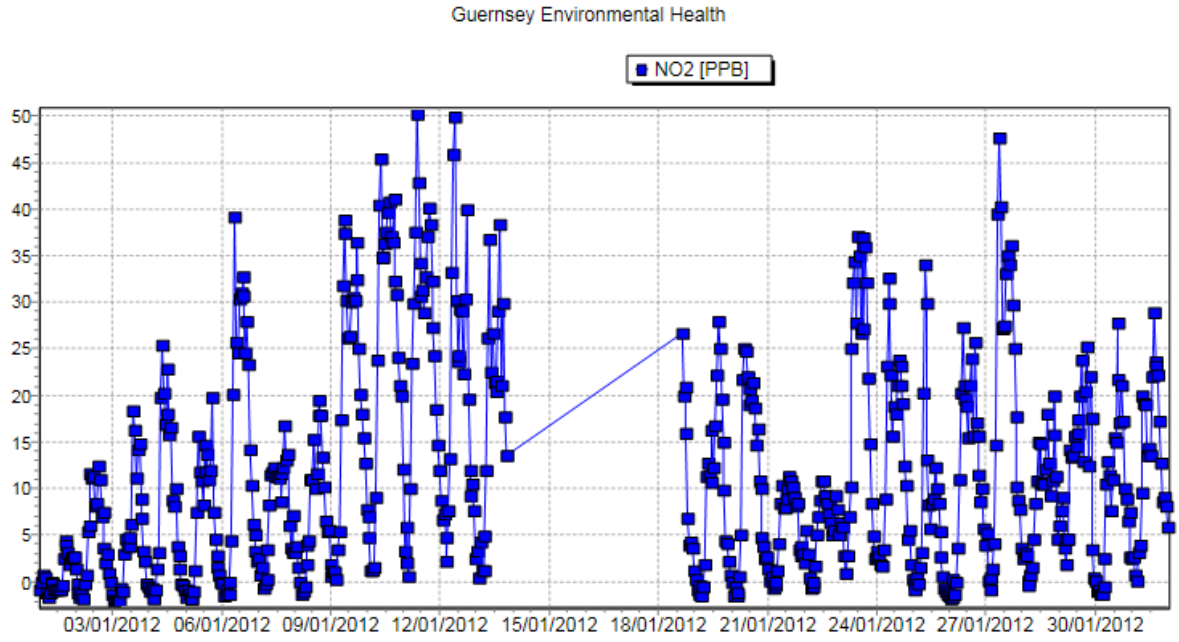
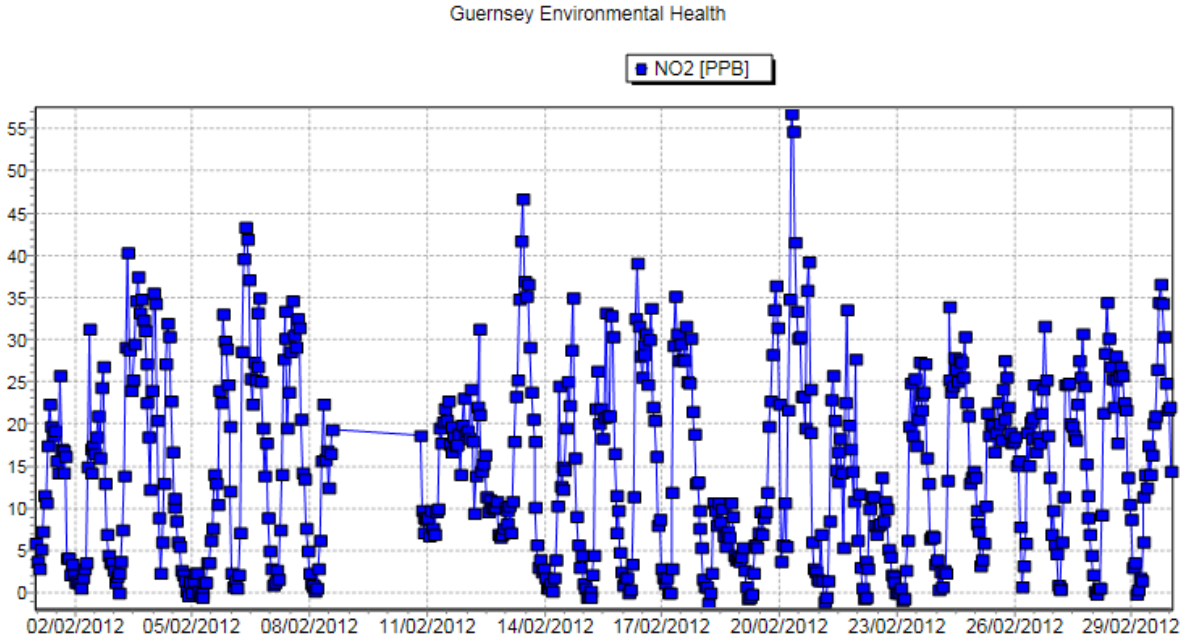
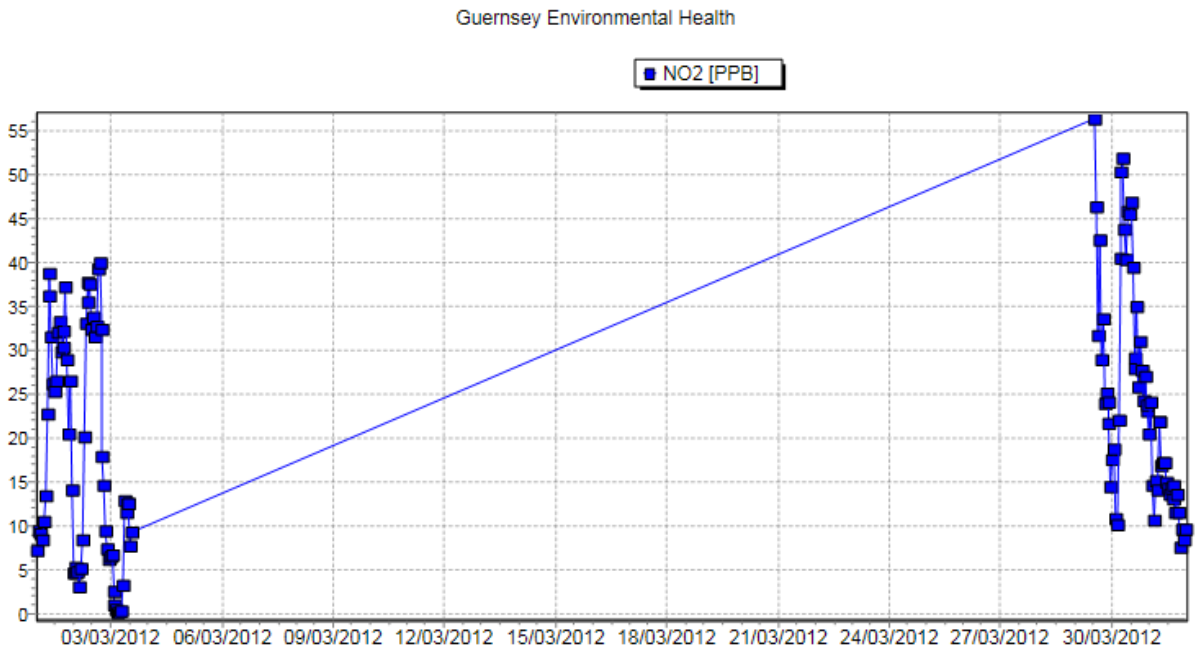
NO_2 data for 2012 – Lukis House, The Grange



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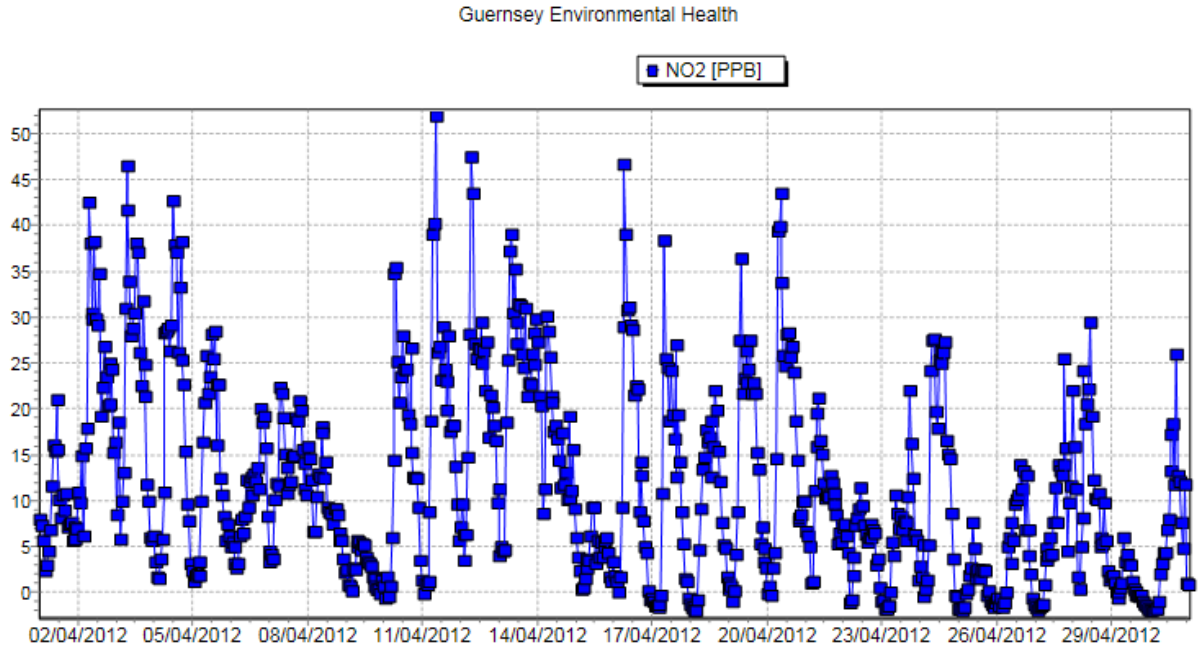
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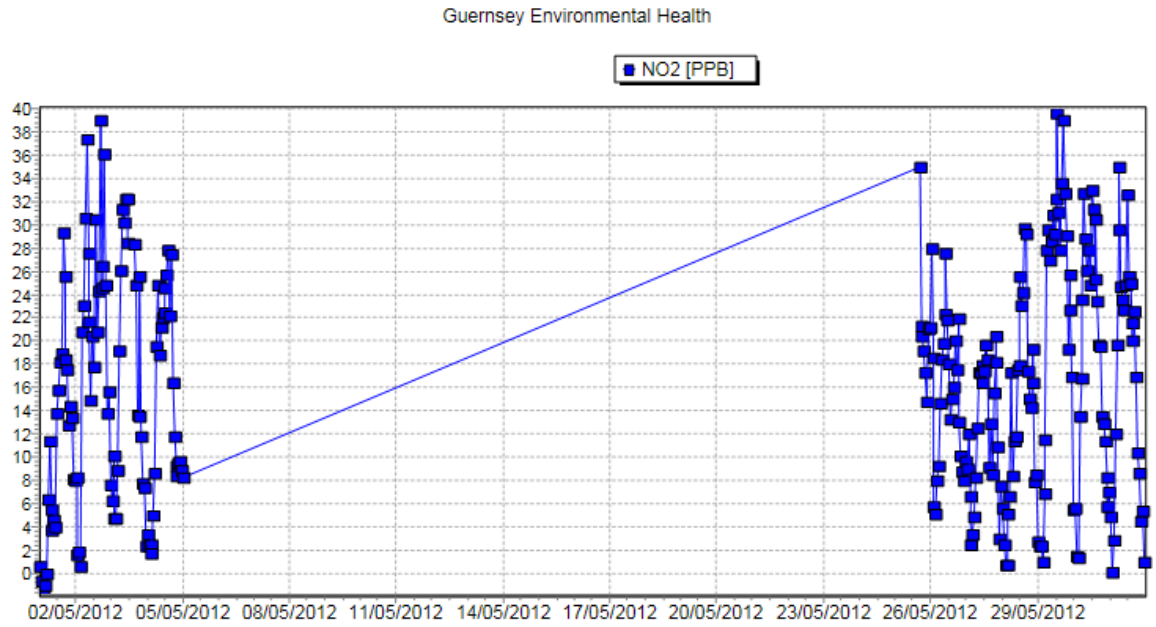
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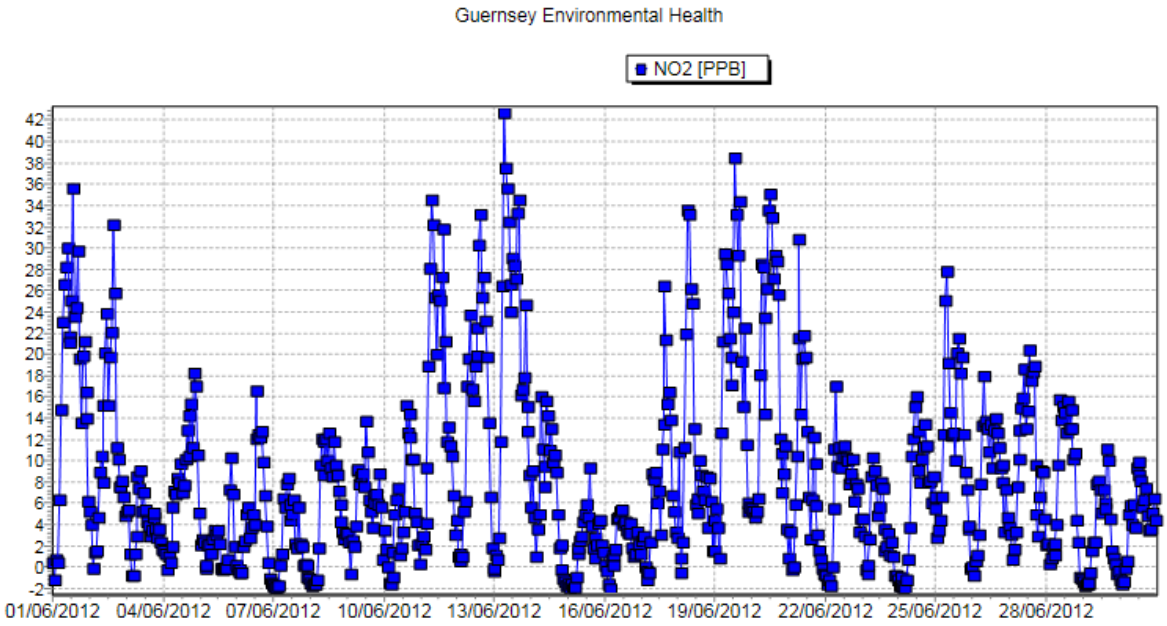
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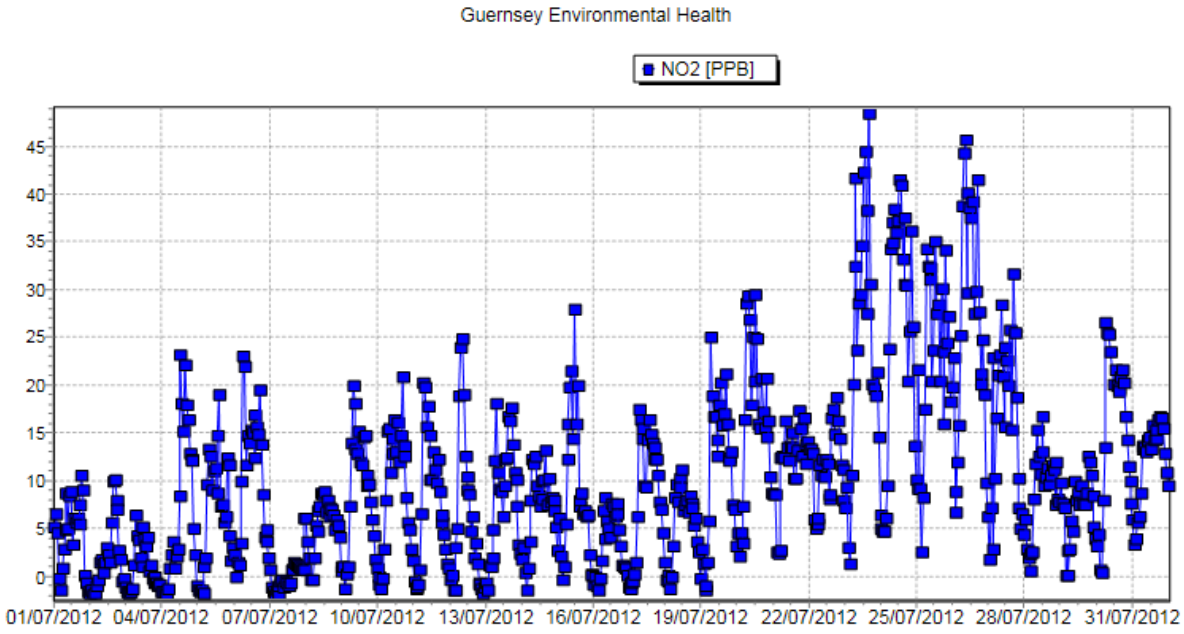
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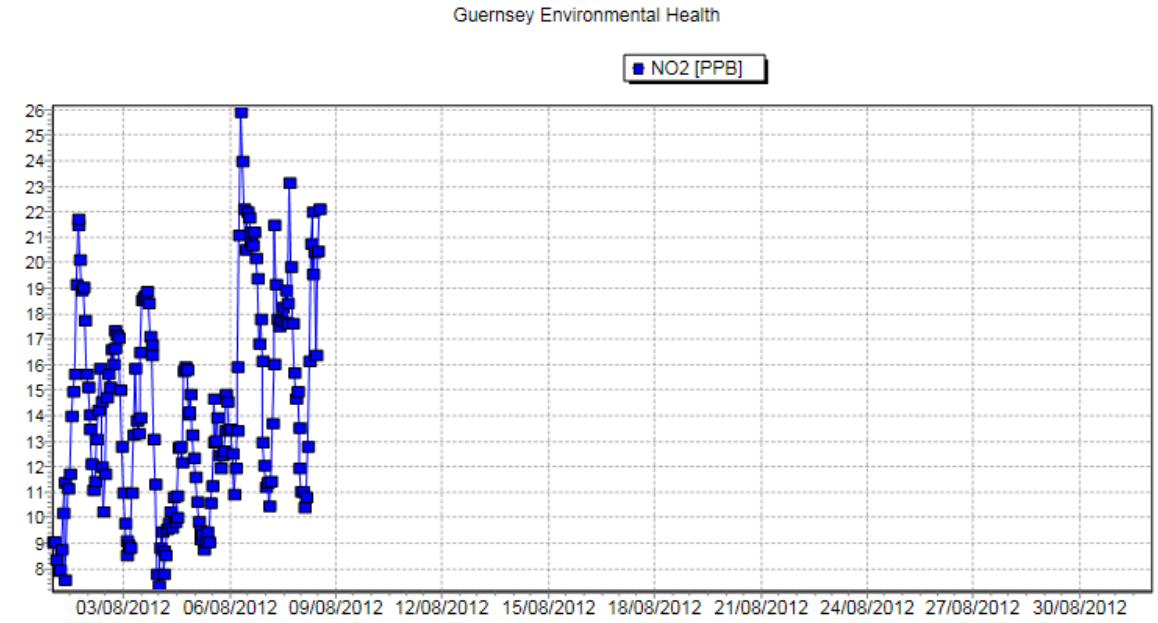
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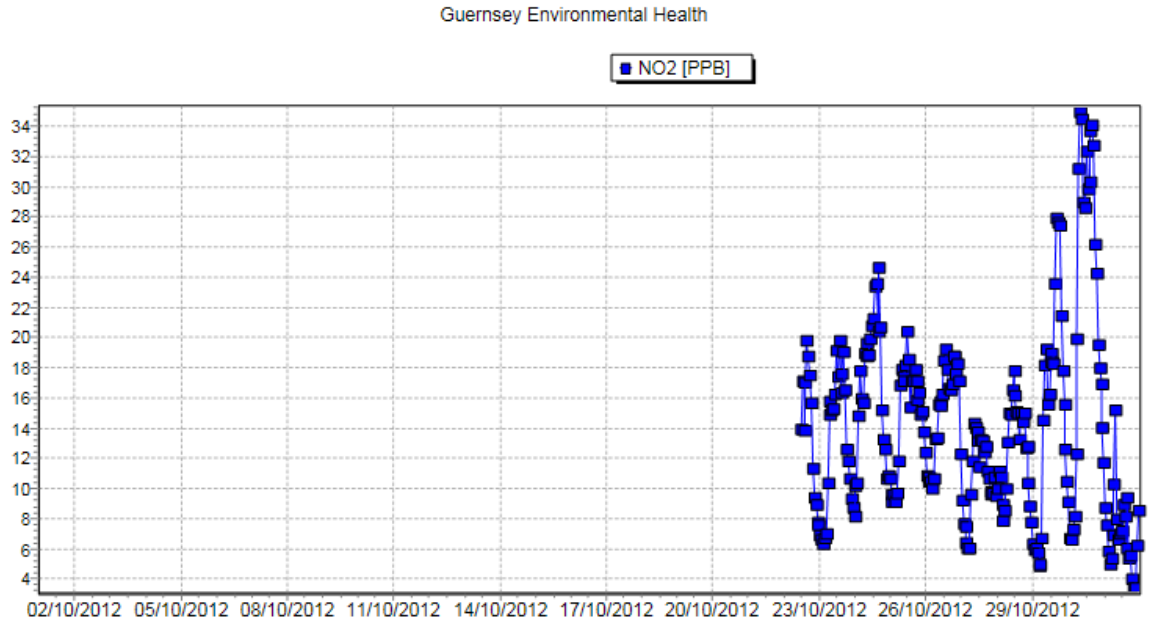
30/08/2012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02/10/2012
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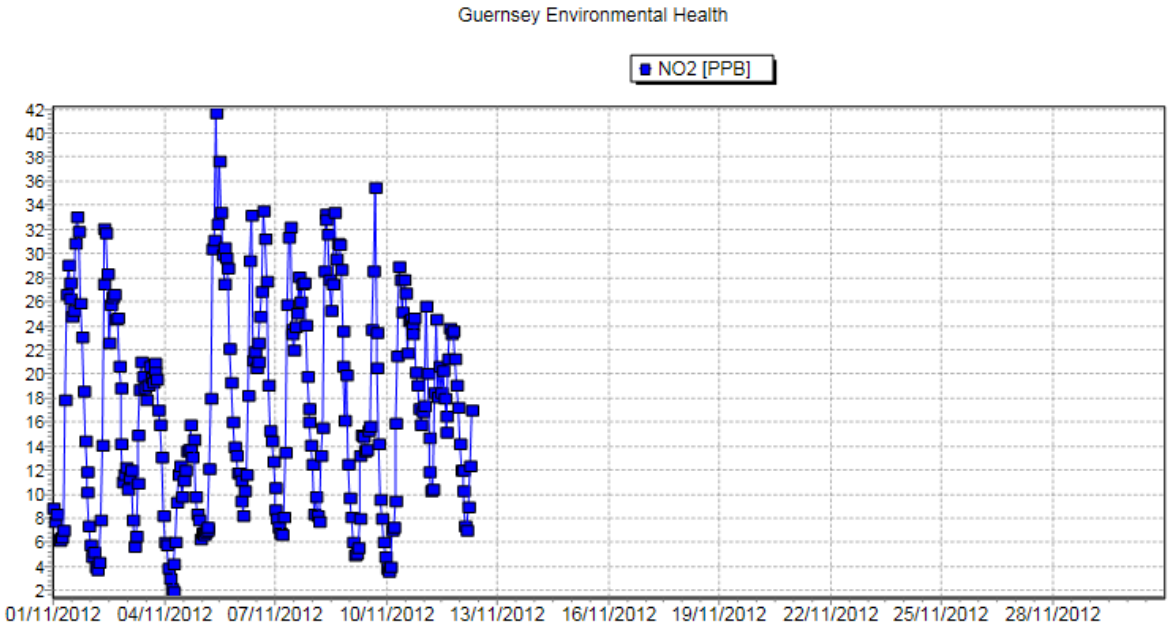
26/10/2012
29/10/2012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01/11/2012
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07/11/2012
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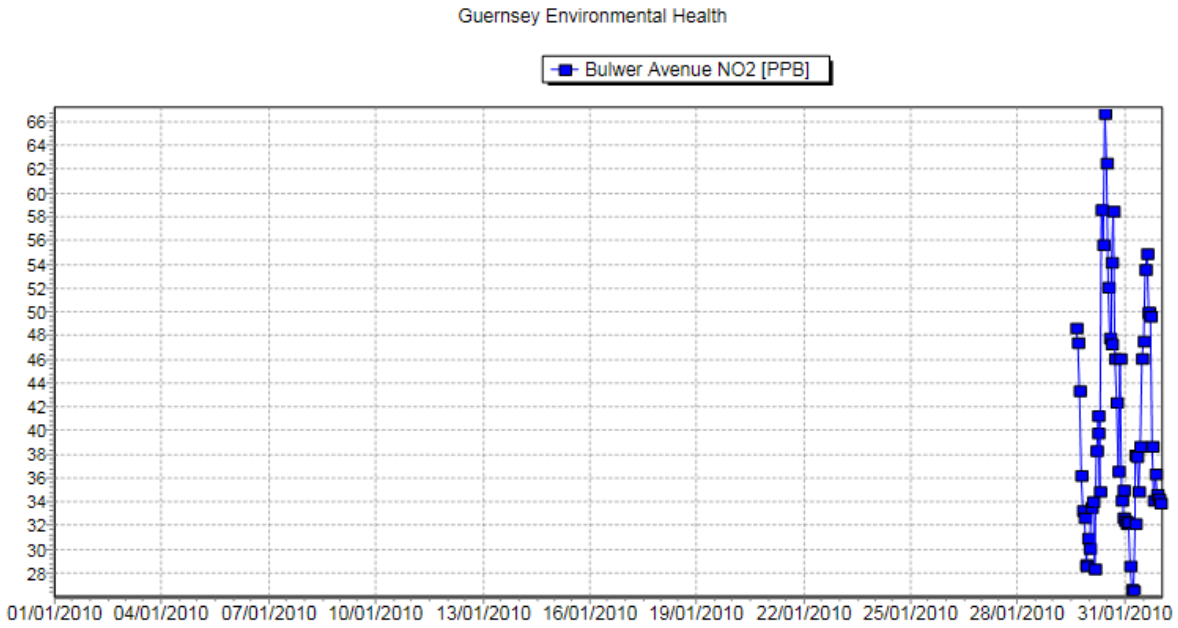
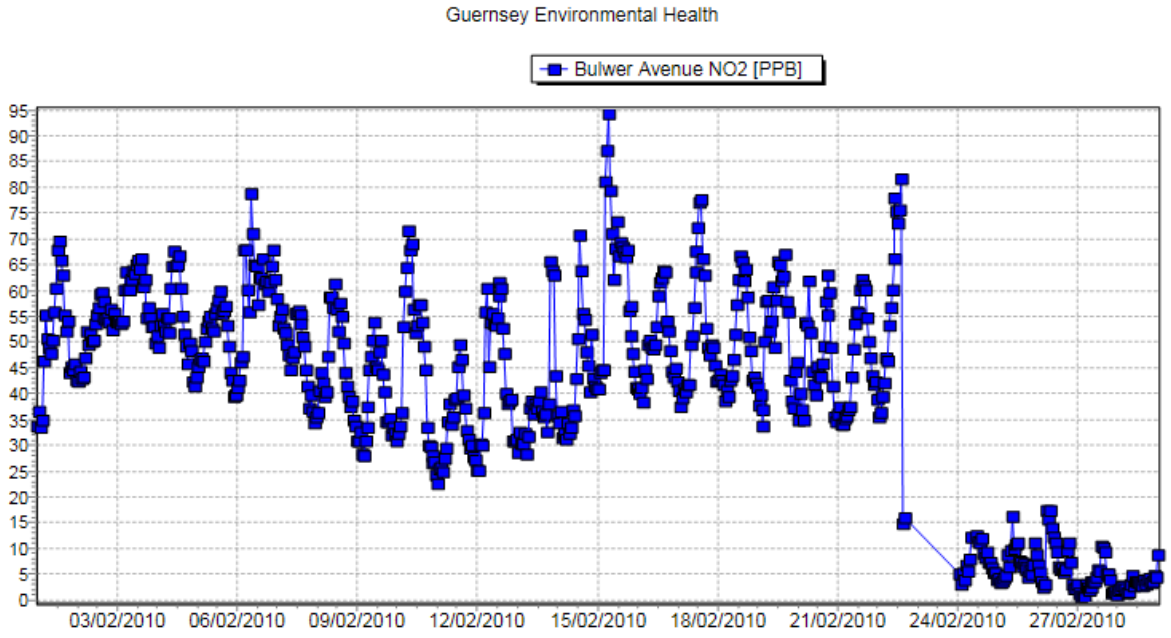
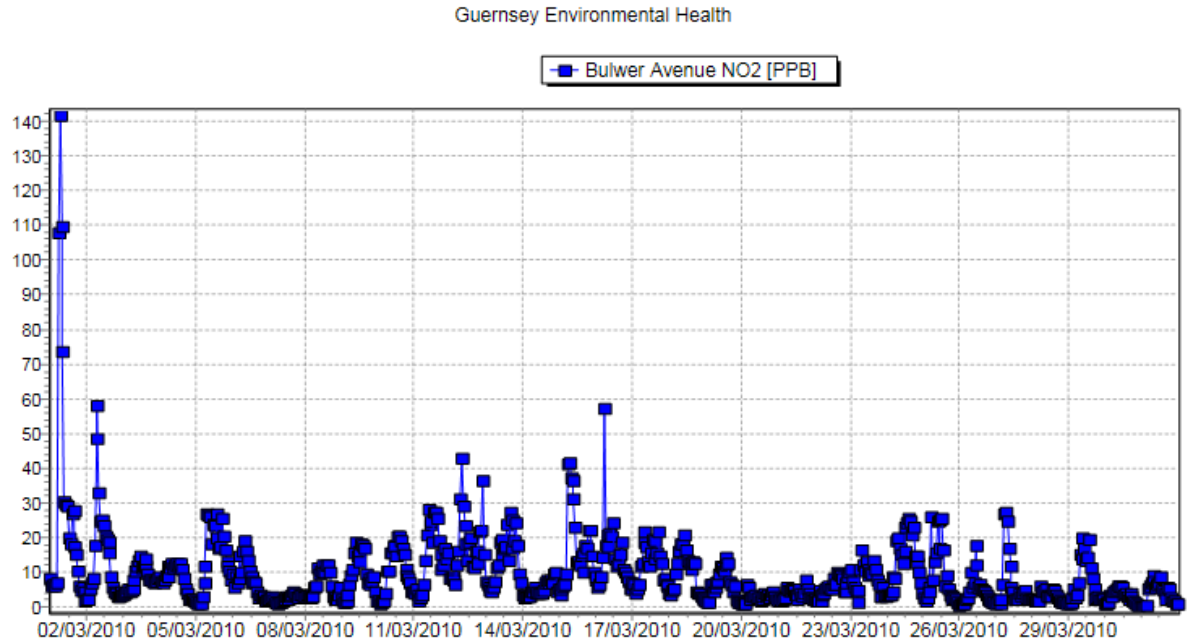
28/11/2012

NO_2 data for 2010 – Bulwer Avenue



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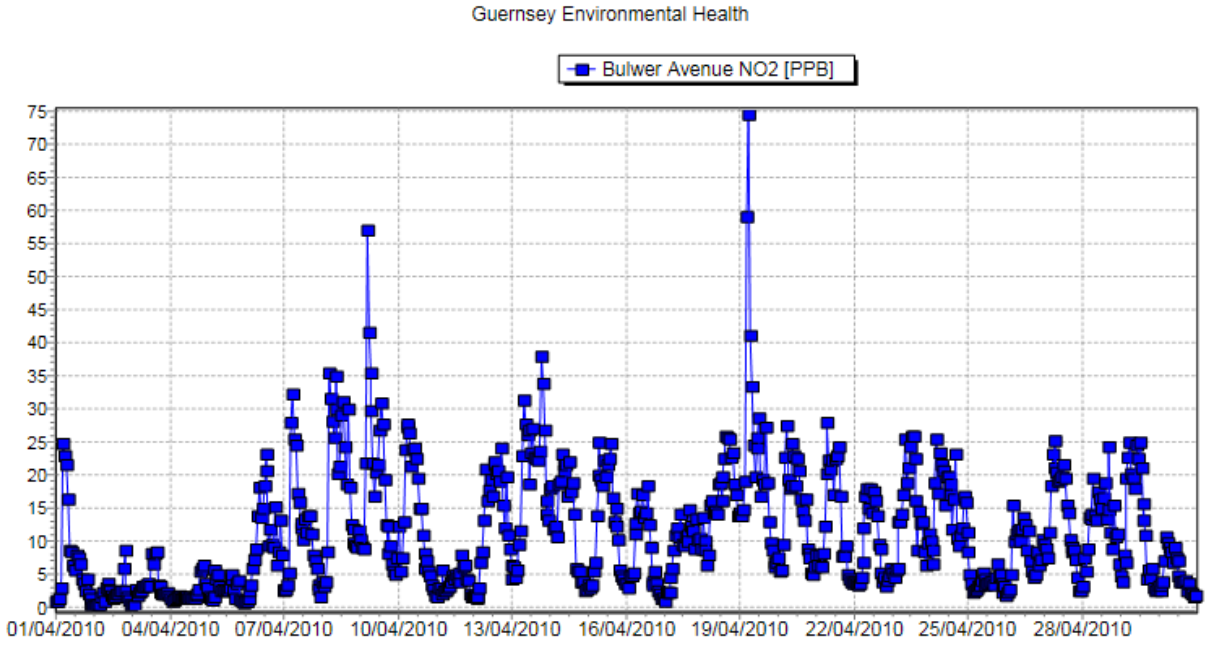
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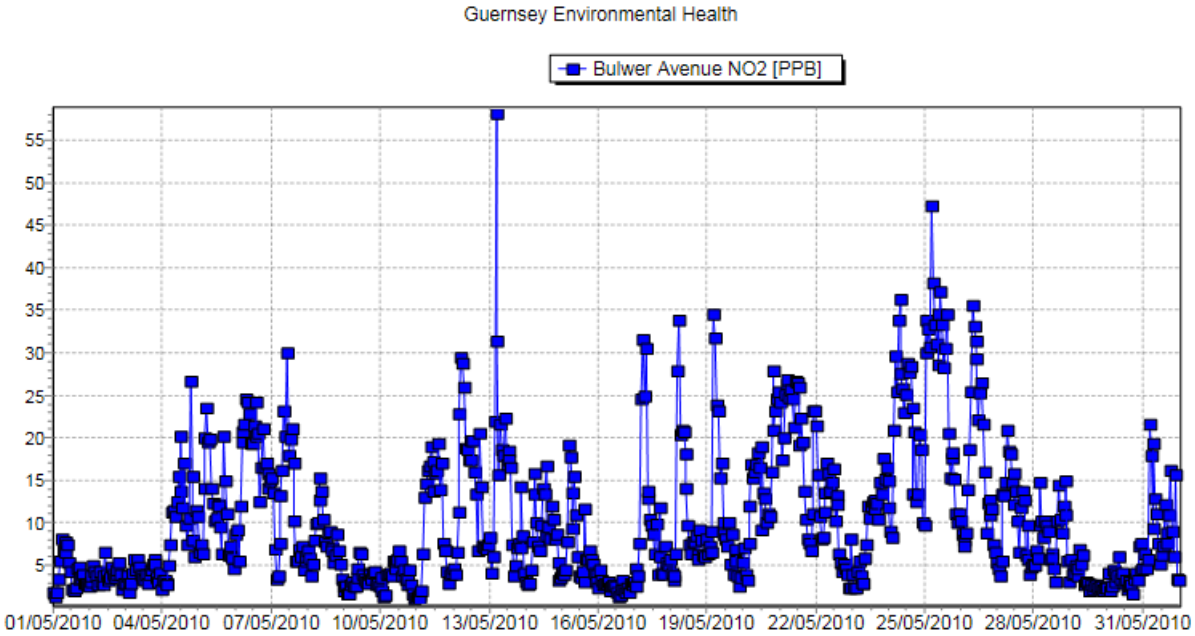
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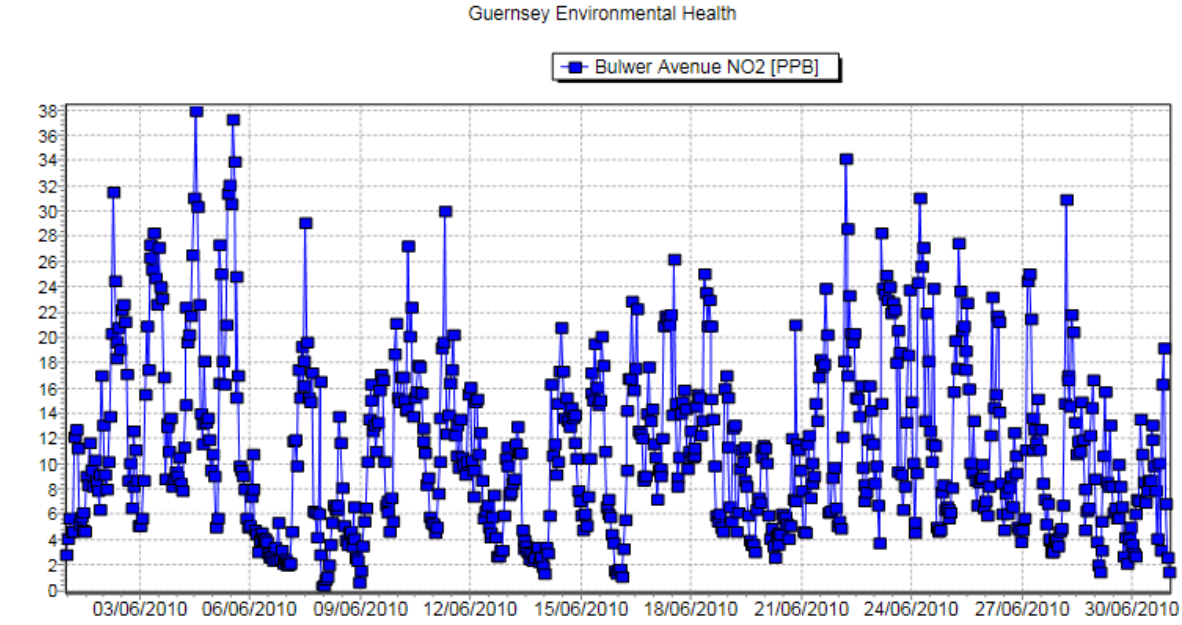
31/05/2010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Bulwer Avenue NO2 [PPB]
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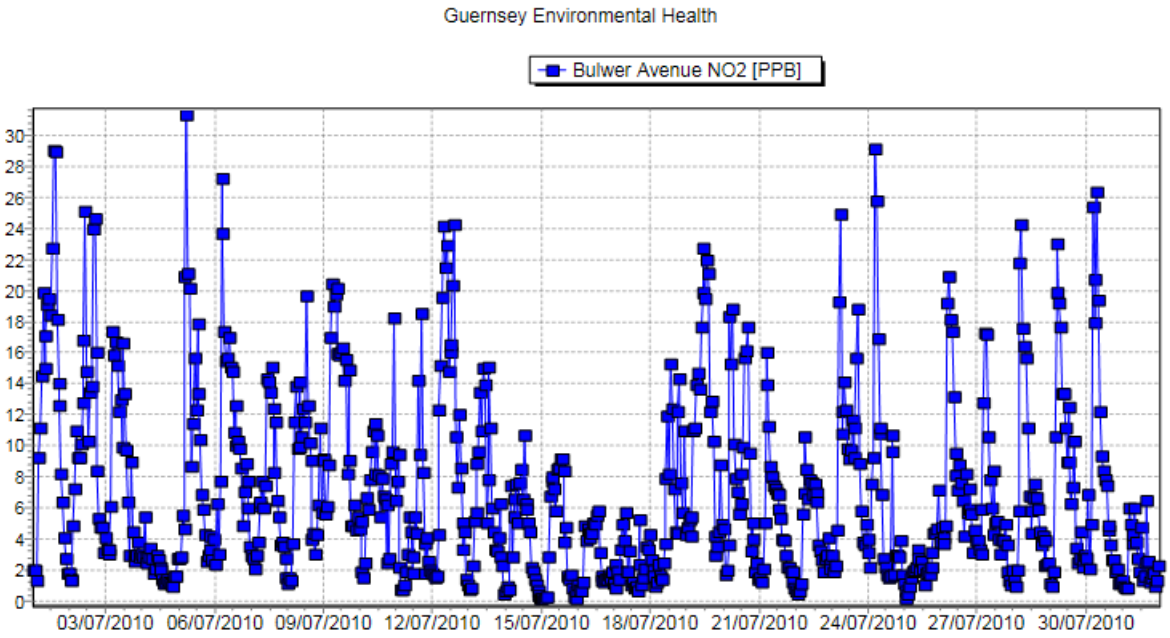
Guernsey Environmental Health
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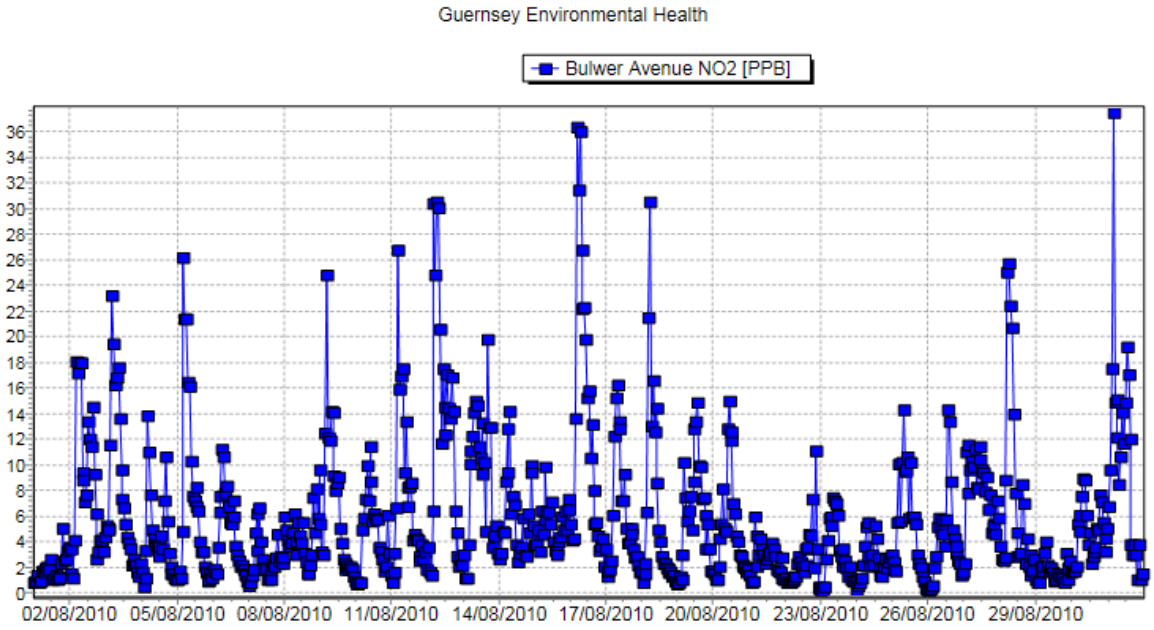
Guernsey Environmental Health
Bulwer Avenue NO2 [PPB]
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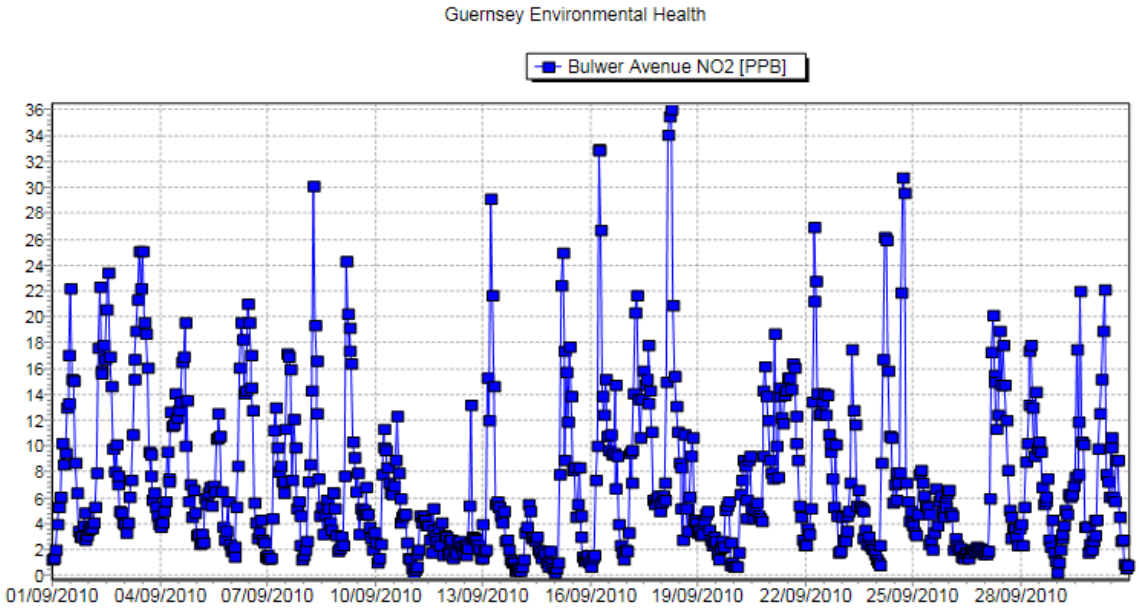
26/08/2010
29/08/2010


Guernsey Environmental Health
Bulwer Avenue NO2 [PPB]
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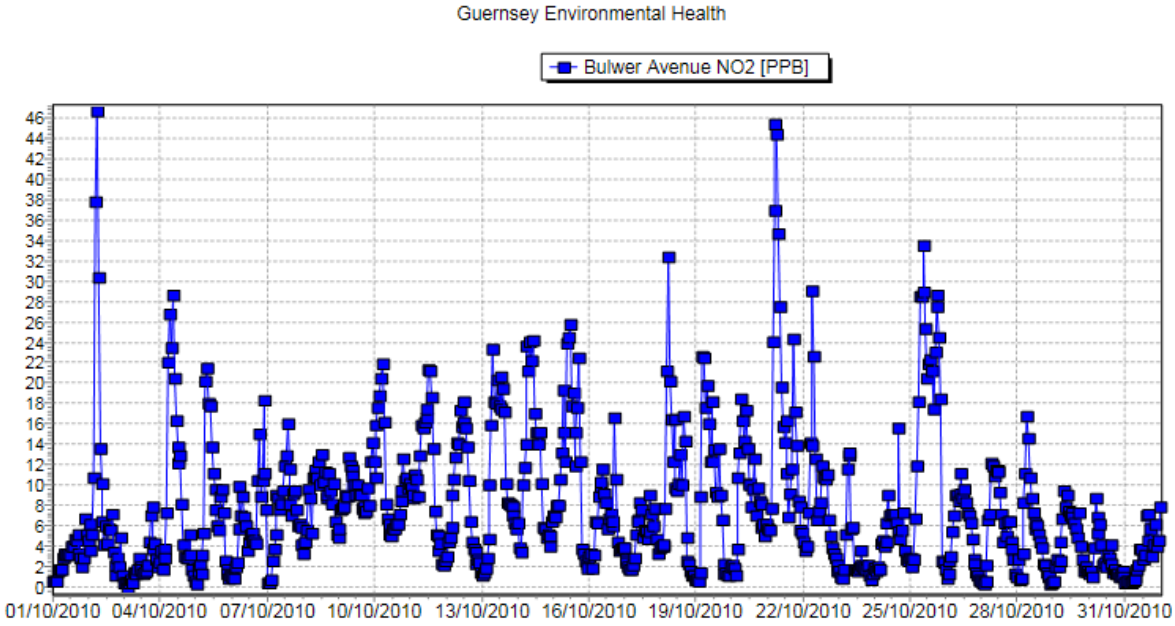
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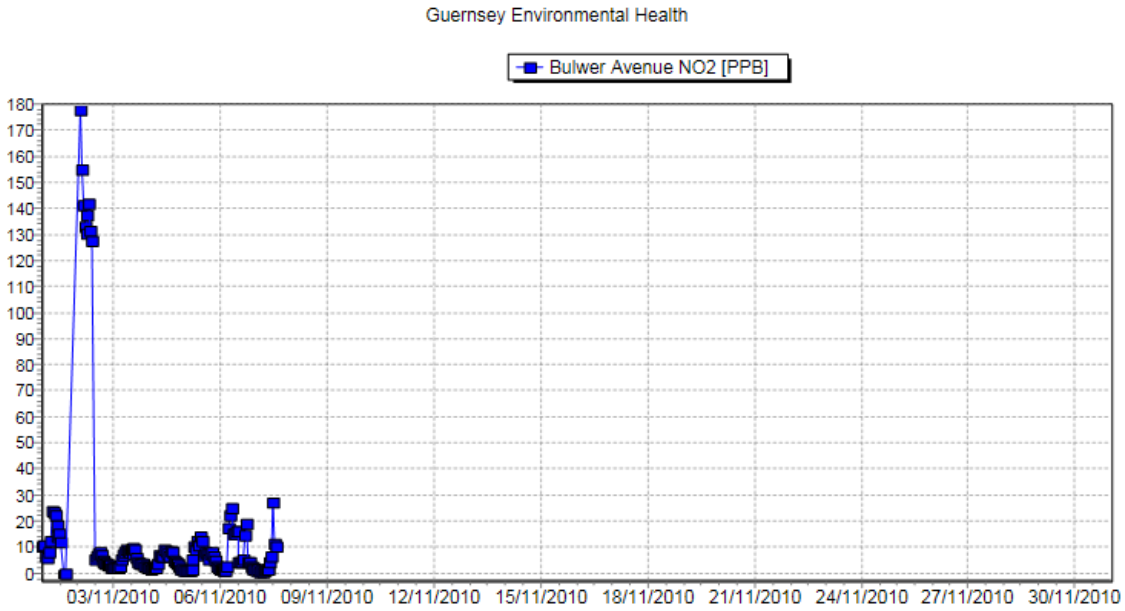

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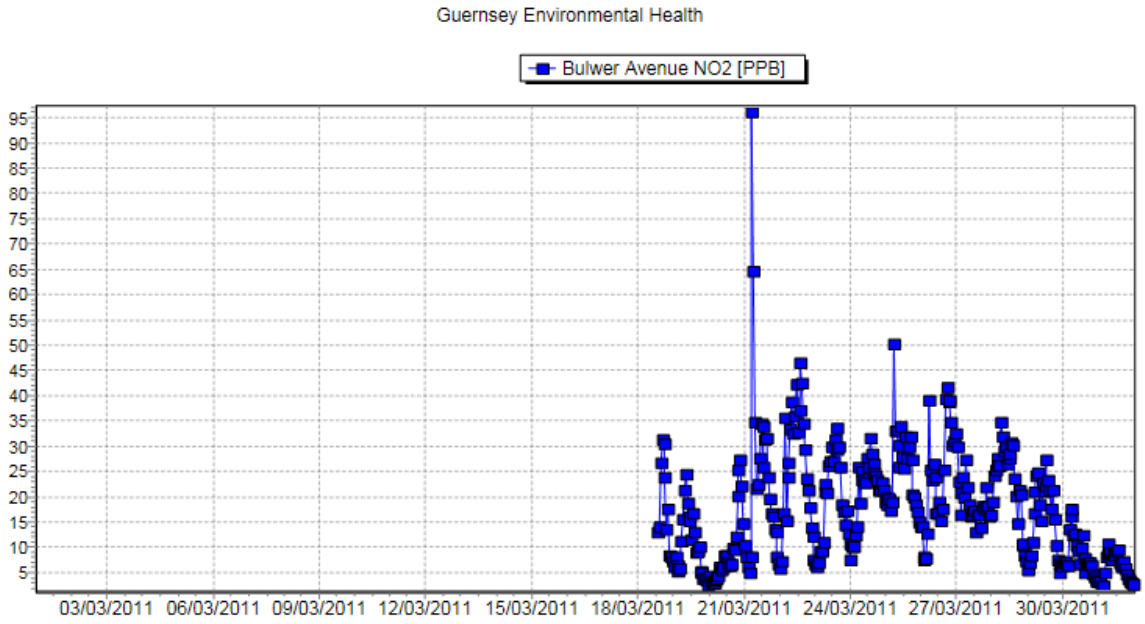
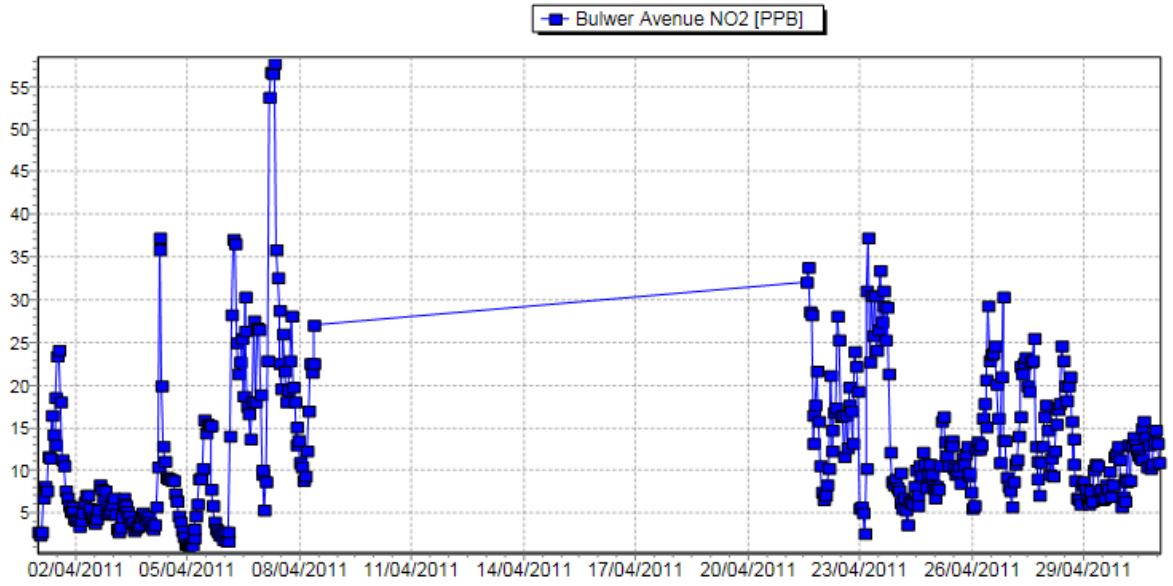
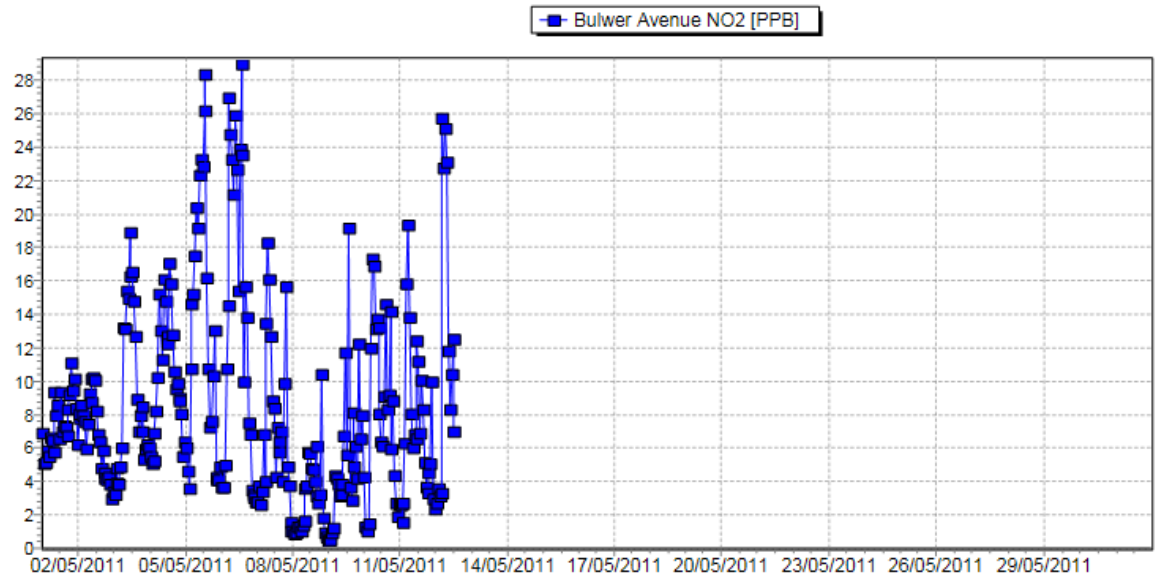
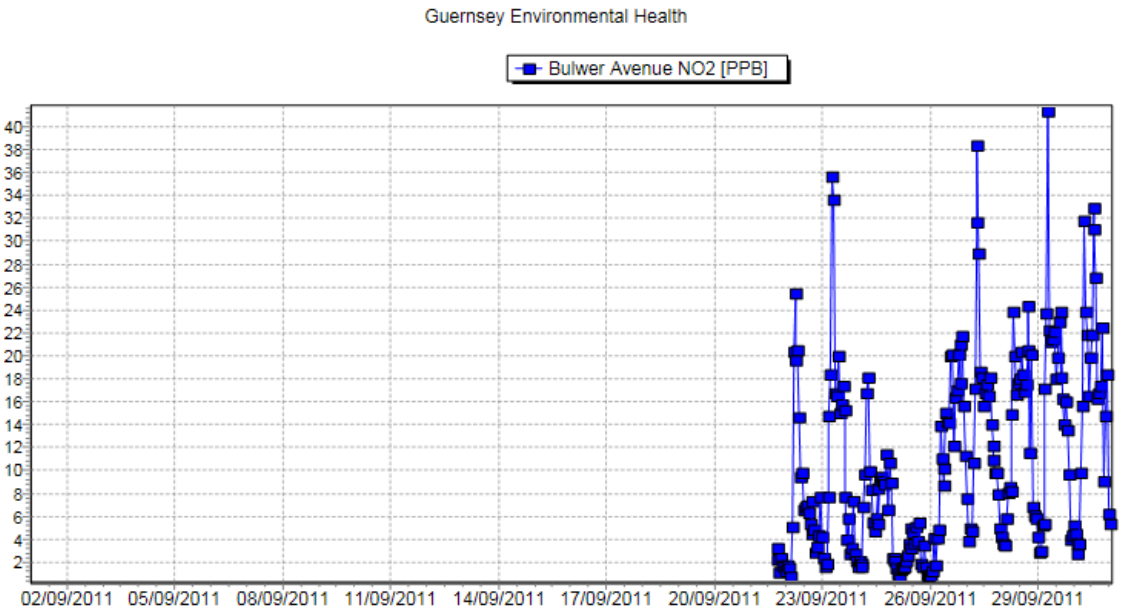


Guernsey Environmental Health



Guernsey Environmental Health




Guernsey Environmental Health
Bulwer Avenue NO2 [PPB]
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02/09/2011
05/09/2011
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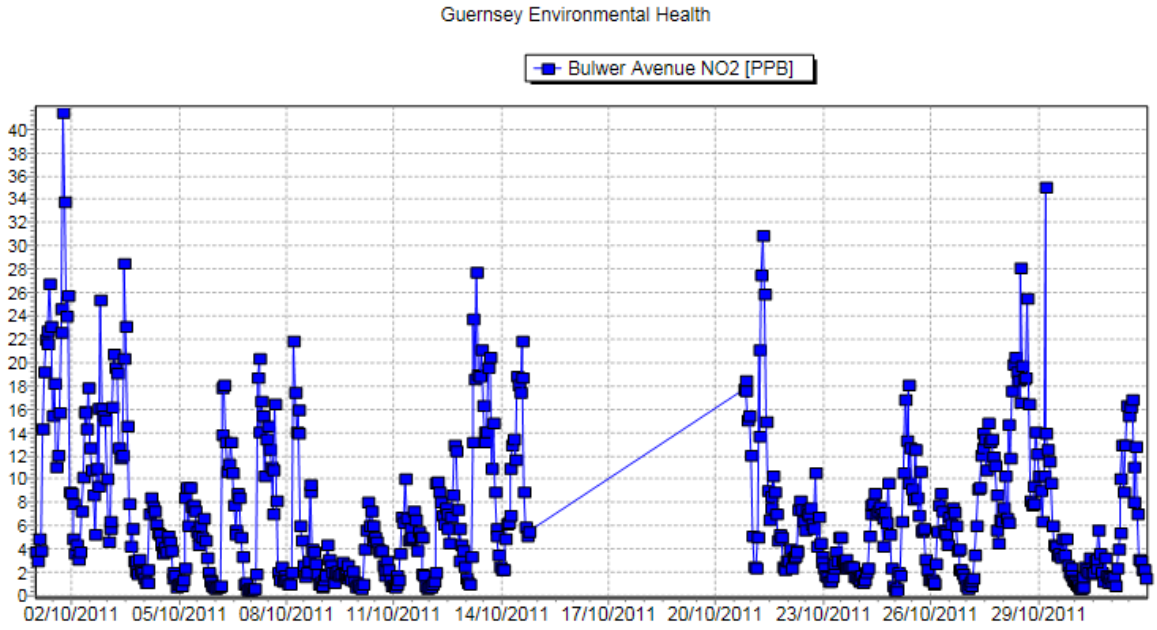
23/09/2011
26/09/2011
29/09/2011


Guernsey Environmental Health
Bulwer Avenue NO2 [PPB]
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02/10/2011
05/10/2011
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17/10/2011
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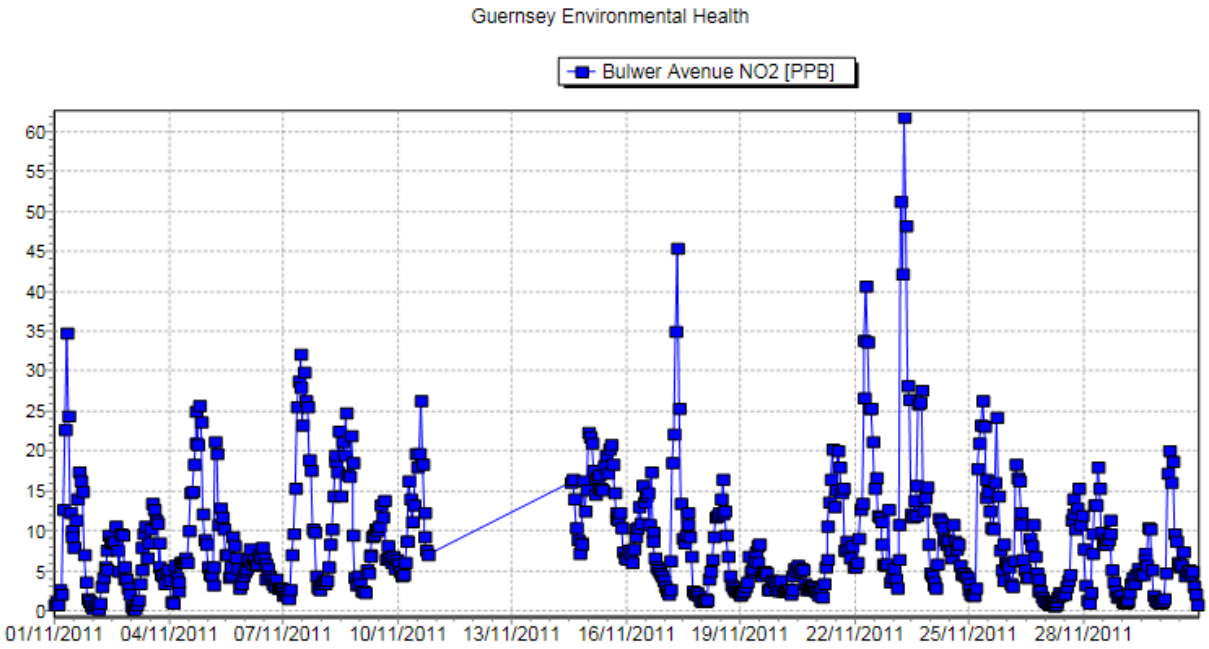
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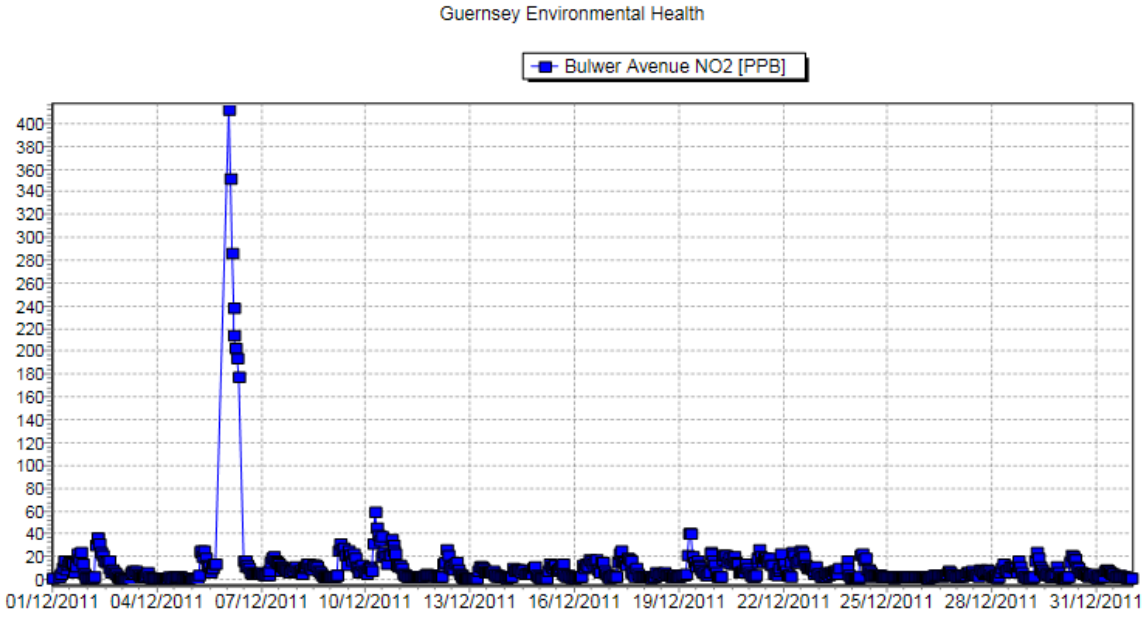
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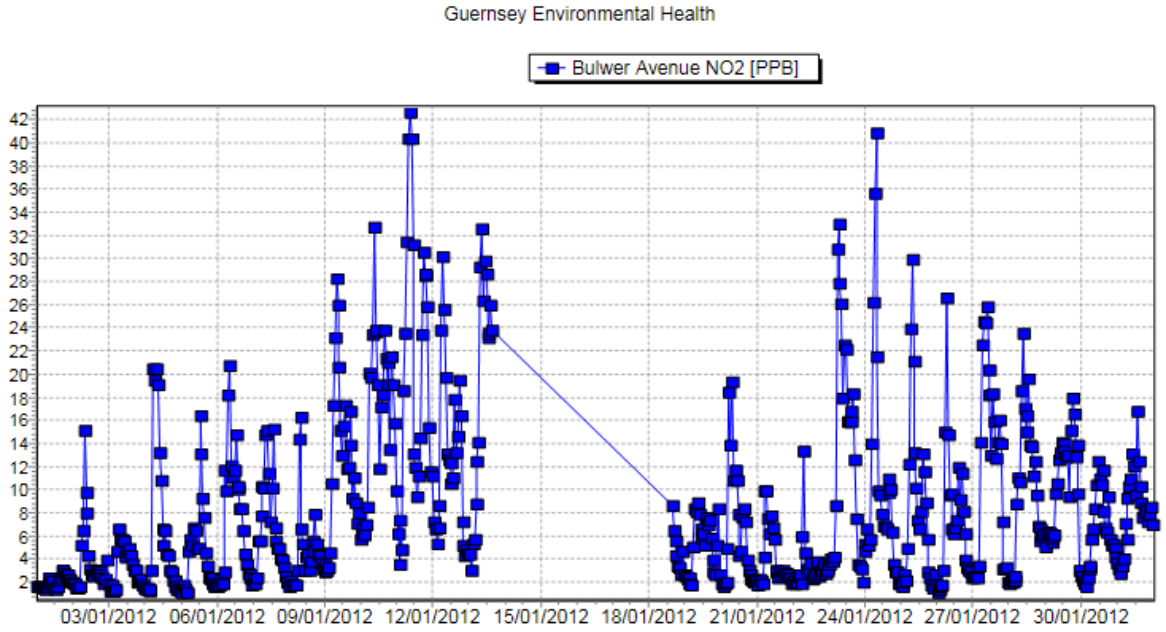
Guernsey Environmental Health
Bulwer Avenue NO2 [PPB]
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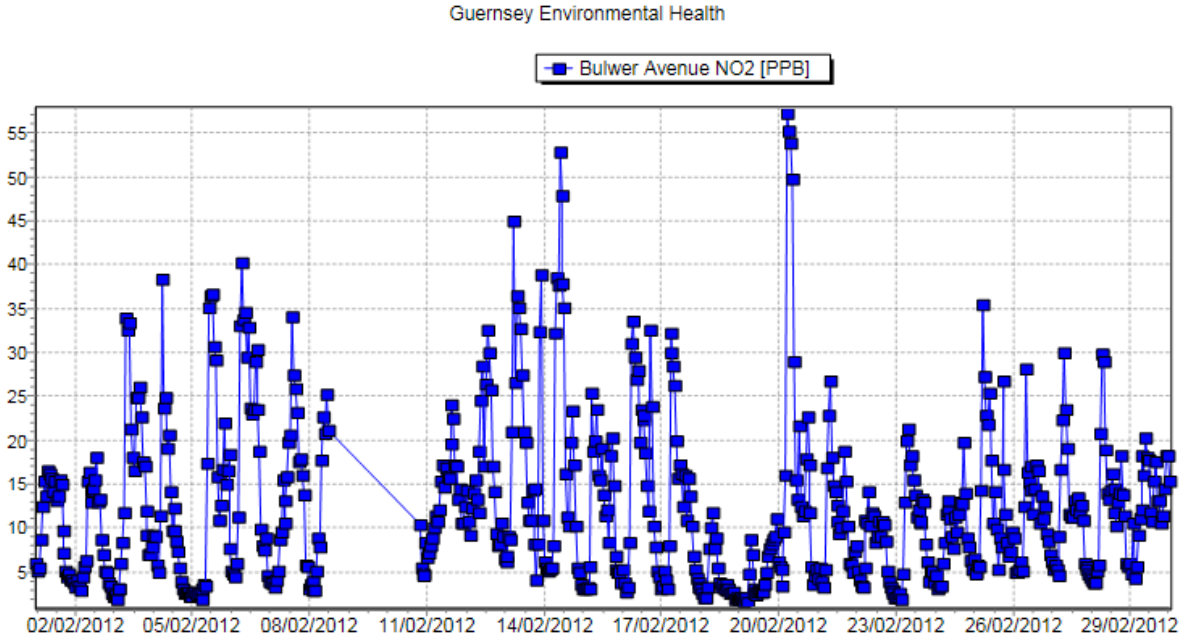
Guernsey Environmental Health
Bulwer Avenue NO2 [PPB]
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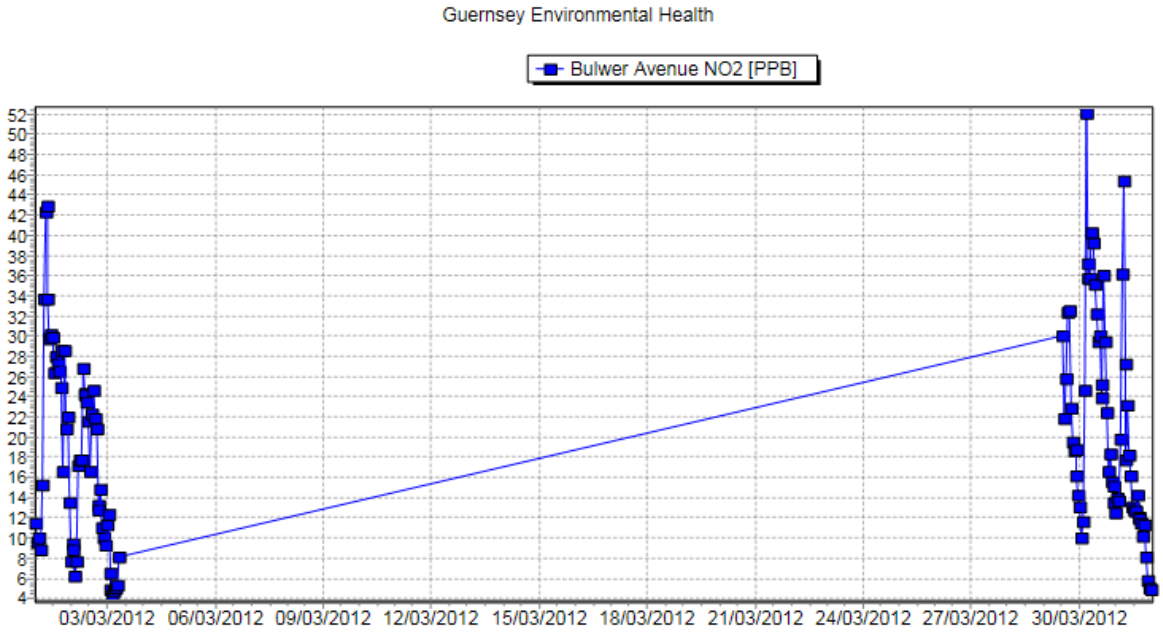
Guernsey Environmental Health
Bulwer Avenue NO2 [PPB]
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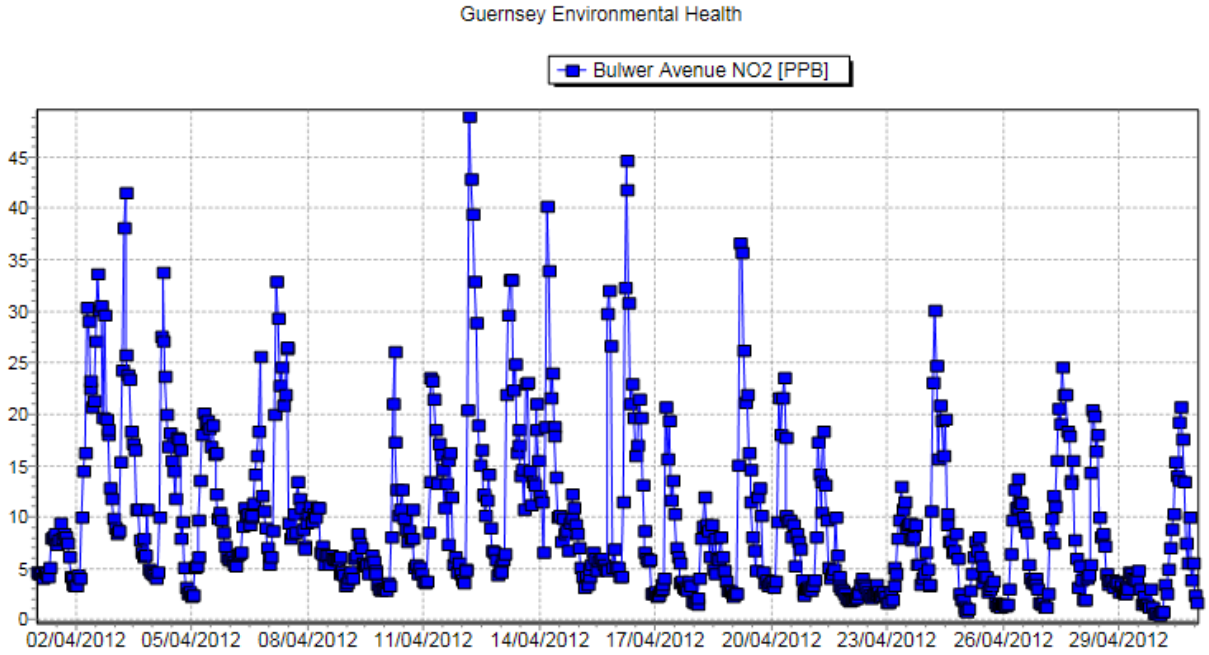
Guernsey Environmental Health
Bulwer Avenue NO2 [PPB]
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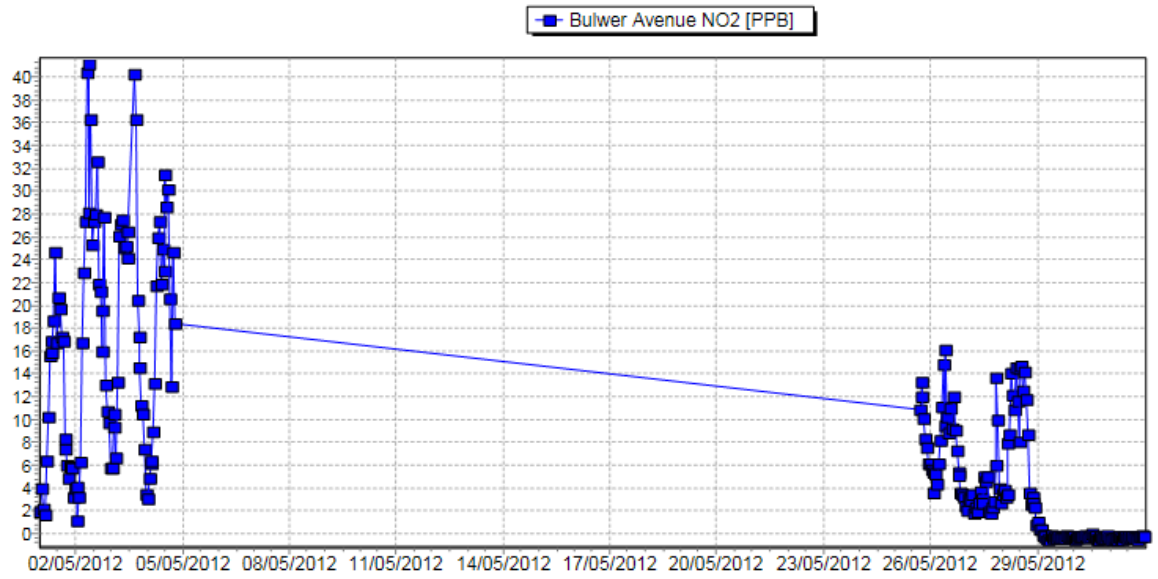
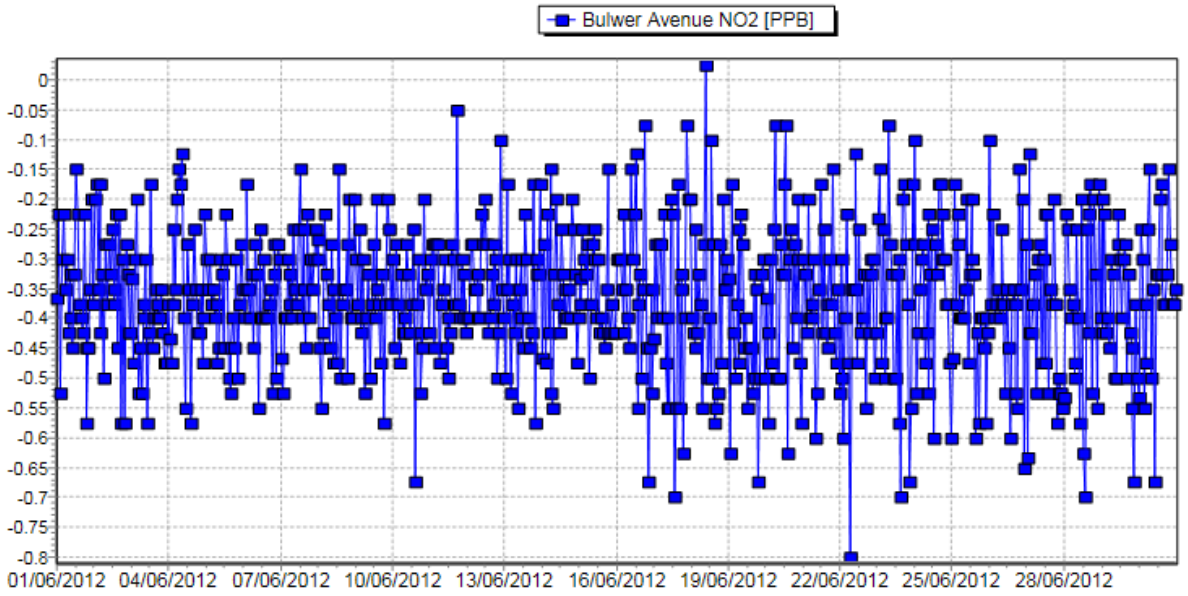
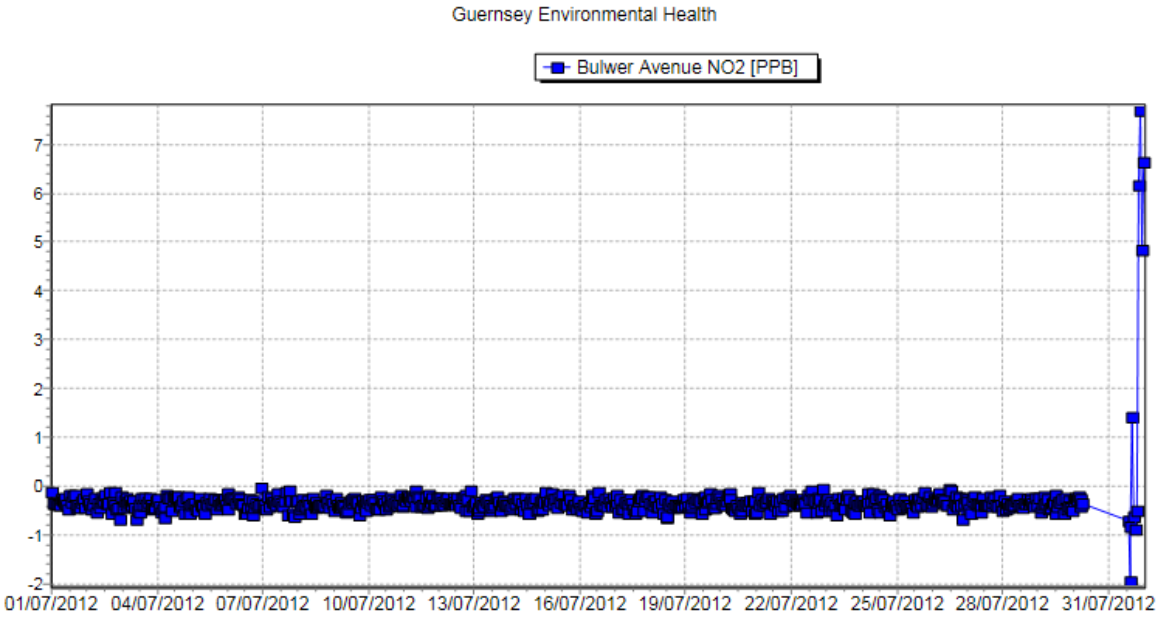
29/04/2012

Guernsey Environmental Health



Guernsey Environmental Health

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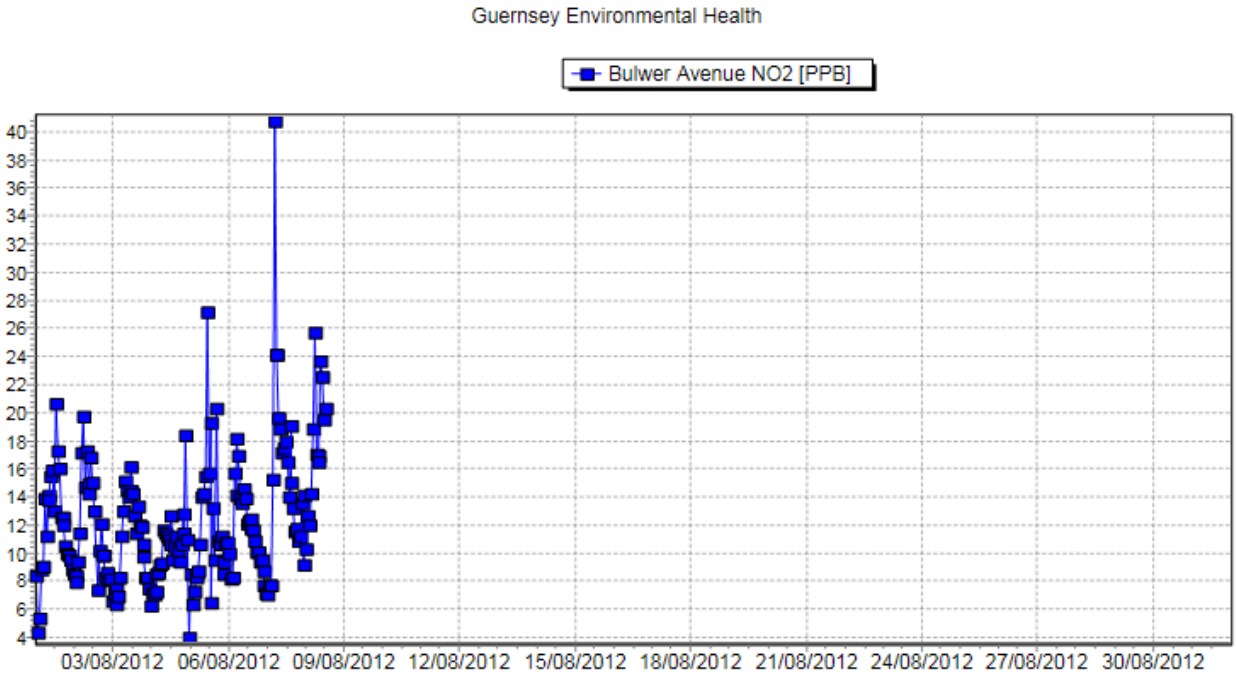
Guernsey Environmental Health
Bulwer Avenue NO2 [PPB]
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12/08/2012
15/08/2012
18/08/2012
21/08/2012
24/08/2012
27/08/2012
30/08/2012

A3.4 Hospital admissions data

Date of admission	Gender	Age	PARISH	DOB
08/01/2008	Male	3	St Peter Port	10/10/2005
20/01/2008	Male	4	Torteval	16/09/2004
01/02/2008	Male	83	St Sampsons	01/09/1925
01/02/2008	Female	3	St Peter Port	03/01/2005
05/02/2008	Female	2	Castel	08/05/2006
11/02/2008	Male	4	St Peter Port	24/02/2004
18/02/2008	Male	9	Vale	08/09/1999
19/02/2008	Female	43	Vale	23/11/1965
19/02/2008	Female	3	St Peter Port	04/03/2005
24/02/2008	Female	2	Castel	08/05/2006
14/03/2008	Female	26	Vale	30/06/1982
15/03/2008	Female	46	St Sampsons	20/04/1962
25/03/2008	Male	1	St Sampsons	02/07/2007
27/03/2008	Female	12	St Peter Port	10/09/1996
07/04/2008	Female	50	Vale	17/02/1958
07/04/2008	Female	4	St Martins	15/04/2004
09/04/2008	Female	33	Castel	26/04/1975
09/04/2008	Male	4	St Sampsons	27/07/2004
11/04/2008	Male	1	Vale	18/03/2007
26/04/2008	Male	41	St Sampsons	31/08/1967
03/05/2008	Male	4	Castel	01/09/2004
06/05/2008	Male	3	St Peter Port	10/10/2005
12/05/2008	Female	39	Vale	19/08/1969
14/05/2008	Male	3	Castel	30/09/2005
18/05/2008	Female	1	Vale	10/04/2007
20/05/2008	Female	2	Castel	19/04/2006
23/05/2008	Male	12	St Martins	20/08/1996
26/05/2008	Female	3	St Peter Port	03/01/2005
12/06/2008	Female	4	St Peter Port	17/07/2004
17/06/2008	Male	11	St Peter Port	09/08/1997
12/07/2008	Female	84	St Andrews	22/06/1924
14/07/2008	Female	3	Castel	05/04/2005
15/07/2008	Female	84	St Andrews	22/06/1924
27/07/2008	Female	18	St Andrews	27/02/1990
08/08/2008	Male	2	St Peters	05/02/2006
20/08/2008	Female	15	St Sampsons	04/09/1993
25/08/2008	Female	84	St Andrews	22/06/1924
28/08/2008	Female	1	Castel	21/08/2007
29/08/2008	Female	2	St Peter Port	24/09/2006
31/08/2008	Male	2	St Sampsons	13/04/2006
03/09/2008	Female	4	St Saviours	27/10/2004
07/09/2008	Female	3	Castel	05/04/2005
08/09/2008	Female	2	Castel	08/05/2006

10/09/2008	Female	7	St Saviours	22/06/2001
12/09/2008	Male	2	St Sampsons	04/10/2006
13/09/2008	Female	10	St Peter Port	07/10/1998
13/09/2008	Male	8	Torteval	26/06/2000
15/09/2008	Male	12	St Martins	20/08/1996
15/09/2008	Male	10	St Sampsons	09/09/1998
15/09/2008	Male	2	St Sampsons	13/04/2006
18/09/2008	Male	11	St Peter Port	09/08/1997
20/09/2008	Male	5	Castel	05/10/2003
22/09/2008	Female	63	St Peter Port	07/11/1945
02/10/2008	Female	4	Vale	27/11/2004
03/10/2008	Male	0	St Peter Port	24/01/2008
14/10/2008	Female	1	Castel	21/08/2007
16/10/2008	Female	3	St Martins	12/04/2005
17/10/2008	Male	1	St Sampsons	18/10/2007
19/10/2008	Female	2	St Peter Port	24/09/2006
20/10/2008	Female	2	Castel	08/05/2006
23/10/2008	Male	1	St Sampsons	28/04/2007
26/10/2008	Female	18	St Andrews	27/02/1990
28/10/2008	Male	3	Castel	30/09/2005
30/10/2008	Male	27	St Sampsons	23/07/1981
31/10/2008	Female	0	Forest	06/03/2008
01/11/2008	Male	0	St Sampsons	24/03/2008
03/11/2008	Female	8	St Peter Port	06/10/2000
09/11/2008	Male	7	St Martins	09/08/2001
19/11/2008	Female	7	Vale	28/05/2001
23/11/2008	Female	2	St Peter Port	19/02/2006
24/11/2008	Male	53	St Peter Port	23/11/1955
26/11/2008	Male	10	St Peter Port	08/08/1998
27/11/2008	Male	58	Vale	02/09/1950
02/12/2008	Female	18	St Andrews	27/02/1990
09/12/2008	Female	32	St Martins	12/11/1977
15/12/2008	Female	63	St Peter Port	27/03/1945
16/12/2008	Male	42	St Sampsons	07/04/1966
17/12/2008	Female	1	Castel	21/08/2007
12/01/2009	Female	49	St Sampsons	24/07/1960
13/01/2009	Male	4	St Peter Port	28/03/2005
16/01/2009	Female	32	St Martins	12/11/1977
21/01/2009	Female	4	Vale	31/10/2005
26/01/2009	Female	53	St Sampsons	26/05/1956
04/02/2009	Male	54	St Peter Port	23/11/1955
15/02/2009	Male	1	St Martins	12/06/2008
17/02/2009	Male	61	St Sampsons	14/07/1948
17/02/2009	Male	43	St Peter Port	28/08/1966
19/02/2009	Male	3	St Sampsons	13/04/2006
26/02/2009	Female	13	St Peter Port	18/08/1996

27/02/2009	Female	6	St Saviours	04/07/2003
06/03/2009	Female	6	St Sampsons	05/09/2003
12/03/2009	Male	4	St Peter Port	28/03/2005
17/03/2009	Male	34	St Peter Port	13/11/1975
25/03/2009	Female	11	St Peter Port	30/05/1998
28/03/2009	Female	71	Torteval	16/07/1938
28/03/2009	Male	2	Vale	03/11/2007
02/04/2009	Male	1	Castel	13/03/2008
05/04/2009	Female	3	St Peter Port	19/02/2006
07/05/2009	Male	2	St Martins	19/12/2007
20/05/2009	Male	7	Vale	10/09/2002
30/05/2009	Female	1	Castel	22/05/2008
03/06/2009	Female	42	St Peter Port	12/10/1967
08/06/2009	Male	54	St Peter Port	23/11/1955
09/06/2009	Male	3	St Peter Port	02/01/2006
21/06/2009	Male	6	Vale	19/05/2003
22/06/2009	Male	3	St Sampsons	04/10/2006
29/06/2009	Male	1	Castel	21/06/2008
09/07/2009	Male	10	Vale	10/08/1999
27/07/2009	Male	5	Castel	05/02/2004
12/08/2009	Male	5	Castel	23/09/2004
15/08/2009	Male	5	St Peter Port	25/08/2004
03/09/2009	Female	41	St Peters	11/09/1968
17/09/2009	Male	14	St Saviours	29/03/1995
20/09/2009	Male	3	Castel	07/08/2006
22/09/2009	Male	14	St Peter Port	17/05/1995
22/09/2009	Male	9	Torteval	26/06/2000
24/09/2009	Male	5	St Peter Port	25/08/2004
24/09/2009	Male	4	St Peter Port	10/10/2005
28/09/2009	Female	28	St Sampsons	14/02/1981
02/10/2009	Female	32	St Martins	12/11/1977
02/10/2009	Female	1	Vale	24/08/2008
09/10/2009	Female	12	Castel	14/10/1997
10/10/2009	Male	1	Vale	13/12/2008
11/10/2009	Female	2	Vale	08/12/2007
14/10/2009	Female	7	Vale	14/07/2002
17/10/2009	Male	13	St Martins	20/08/1996
19/10/2009	Female	84	St Peter Port	15/04/1925
21/10/2009	Female	23	Vale	06/10/1986
22/10/2009	Female	4	St Peter Port	08/04/2005
22/10/2009	Female	2	Vale	15/12/2007
31/10/2009	Female	4	St Peter Port	01/06/2005
02/11/2009	Male	0	Vale	13/12/2008
03/11/2009	Female	1	Vale	24/08/2008
08/11/2009	Female	5	St Saviours	06/08/2004
10/11/2009	Male	3	St Sampsons	05/08/2006

13/11/2009	Male	3	St Saviours	21/11/2005
16/11/2009	Male	3	Vale	21/11/2005
19/11/2009	Male	2	Alderney	18/10/2007
25/11/2009	Female	8	Vale	13/11/2001
29/11/2009	Male	46	St Peter Port	06/09/1963
03/12/2009	Female	4	Castel	06/01/2005
09/12/2009	Female	64	Forest	10/09/1945
11/12/2009	Male	52	St Peter Port	07/06/1957
12/12/2009	Male	13	St Martins	20/08/1996
14/12/2009	Male	2	Vale	06/03/2007
19/12/2009	Female	64	St Peter Port	20/11/1945
22/12/2009	Male	1	Vale	13/12/2008
02/01/2010	Female	39	Castel	31/08/1970
05/01/2010	Female	11	St Sampsons	30/05/1998
06/01/2010	Female	19	St Peter Port	02/08/1990
09/01/2010	Female	68	Castel	06/01/1942
15/01/2010	Male	10	St Peter Port	01/04/1999
16/01/2010	Male	4	St Peter Port	28/03/2005
20/01/2010	Female	44	Vale	23/11/1965
21/01/2010	Male	5	St Martins	25/12/2004
14/02/2010	Male	2	St Andrews	24/01/2008
26/02/2010	Male	1	Forest	09/06/2008
15/03/2010	Male	4	St Peter Port	28/03/2005
17/03/2010	Female	73	Castel	26/03/1936
21/03/2010	Male	12	St Peter Port	23/04/1997
28/03/2010	Male	2	Vale	15/05/2007
18/04/2010	Male	2	Herm	28/11/2007
20/04/2010	Female	3	St Saviours	10/04/2007
20/04/2010	Female	1	St Peter Port	05/01/2009
22/04/2010	Female	3	St Saviours	10/04/2007
22/04/2010	Female	3	St Saviours	10/04/2007
01/05/2010	Male	2	Vale	15/05/2007
10/05/2010	Female	18	St Peter Port	20/07/1991
10/05/2010	Male	5	St Peter Port	28/03/2005
18/05/2010	Female	2	St Martins	23/01/2008
26/05/2010	Male	64	No address	25/03/1946
28/05/2010	Female	83	St Peter Port	18/03/1927
03/06/2010	Male	2	St Peter Port	13/05/2008
08/06/2010	Female	65	St Peter Port	15/03/1945
07/07/2010	Male	4	Vale	22/03/2006
23/07/2010	Male	2	Forest	09/06/2008
27/07/2010	Female	20	Vale	25/03/1990
27/07/2010	Female	2	St Martins	23/01/2008
28/07/2010	Female	2	St Sampsons	28/04/2008
25/08/2010	Female	76	St Peter Port	14/04/1934
25/08/2010	Male	2	Sark	18/12/2007

12/09/2010	Male	44	St Martins	23/03/1966
26/09/2010	Female	20	St Andrews	27/02/1990
26/09/2010	Female	7	St Saviours	04/07/2003
26/09/2010	Female	18	St Sampsons	14/05/1992
30/09/2010	Female	38	Castel	15/07/1972
04/10/2010	Male	10	Torteval	26/06/2000
09/10/2010	Female	46	St Martins	29/12/1963
11/10/2010	Female	29	St Peter Port	31/05/1981
26/10/2010	Male	2	St Sampsons	20/12/2007
27/10/2010	Male	4	St Martins	22/02/2006
04/11/2010	Female	44	Vale	23/11/1965
10/11/2010	Male	9	Vale	20/10/2001
19/11/2010	Female	1	St Peter Port	05/01/2009
20/11/2010	Female	3	Vale	12/05/2007
07/12/2010	Male	15	St Saviours	06/11/1995
14/12/2010	Female	76	Castel	23/04/1934
30/12/2010	Male	19	Castel	18/09/1991
31/12/2010	Female	28	Vale	30/06/1982
22/01/2011	Female	72	Castel	02/03/1938
22/01/2011	Male	1	St Peter Port	23/12/2009
24/01/2011	Female	61	St Sampsons	08/05/1949
24/01/2011	Male	5	St Andrews	10/08/2005
18/02/2011	Male	2	Castel	18/02/2009
20/02/2011	Female	4	St Peter Port	09/09/2006
22/02/2011	Male	2	St Peter Port	13/05/2008
24/02/2011	Female	4	St Peter Port	31/12/2006
25/02/2011	Male	0	Vale	07/10/2010
27/02/2011	Male	1	Vale	08/07/2009
02/03/2011	Female	1	St Martins	14/12/2009
07/03/2011	Female	76	Vale	06/12/1934
23/03/2011	Female	9	St Martins	23/10/2001
24/03/2011	Male	1	Castel	29/10/2009
27/03/2011	Male	1	Sark	01/06/2009
30/03/2011	Male	80	Vale	30/09/1930
31/03/2011	Male	38	Vale	09/06/1972
05/04/2011	Male	2	Forest	09/06/2008
06/04/2011	Female	87	Vale	03/01/1924
13/04/2011	Female	2	St Peter Port	18/10/2008
14/04/2011	Male	83	St Peter Port	11/10/1927
15/04/2011	Male	2	Sark	21/01/2009
24/04/2011	Male	46	St Peter Port	20/05/1964
29/04/2011	Male	3	St Sampsons	18/10/2007
01/05/2011	Female	0	St Andrews	02/09/2010
06/05/2011	Female	56	St Sampsons	28/05/1954
31/05/2011	Female	20	St Peter Port	02/08/1990
01/06/2011	Male	2	Forest	09/06/2008

09/06/2011	Male	1	St Sampsons	20/10/2009
15/06/2011	Female	3	Vale	15/12/2007
15/06/2011	Male	2	Castel	20/10/2008
18/06/2011	Male	1	St Peter Port	23/12/2009
21/06/2011	Female	20	St Peter Port	05/10/1990
30/06/2011	Female	5	Castel	08/05/2006
23/07/2011	Female	1	St Martins	04/07/2010
26/07/2011	Female	6	Vale	27/11/2004
05/08/2011	Female	64	Forest	10/12/1946
17/08/2011	Female	21	St Peter Port	02/08/1990
22/08/2011	Male	5	St Peter Port	26/02/2006
23/08/2011	Male	47	St Peter Port	20/05/1964
10/09/2011	Female	2	St Peter Port	18/10/2008
20/09/2011	Female	2	St Peter Port	25/10/2008
22/09/2011	Female	12	St Peter Port	09/09/1999
27/09/2011	Female	29	Vale	04/12/1981
10/10/2011	Female	17	Torteval	19/09/1994
18/10/2011	Male	3	Forest	09/06/2008
22/10/2011	Female	4	St Saviours	10/04/2007
04/11/2011	Female	55	St Sampsons	26/05/1956
12/11/2011	Female	3	St Peter Port	18/10/2008
13/11/2011	Female	20	St Peter Port	03/01/1991
13/11/2011	Female	5	Castel	08/05/2006
14/11/2011	Female	23	St Peter Port	17/11/1987
16/11/2011	Female	21	St Peter Port	02/08/1990
20/11/2011	Female	5	St Peter Port	31/08/2006
24/11/2011	Male	4	St Martins	15/05/2007
03/12/2011	Male	48	St Peter Port	06/09/1963
13/12/2011	Female	60	St Peter Port	09/04/1951
30/12/2011	Female	46	Vale	23/11/1965
10/01/2012	Female	28	St Saviours	14/08/1983
19/01/2012	Female	4	St Saviours	10/04/2007
20/01/2012	Female	21	St Peter Port	02/08/1990
22/01/2012	Male	3	Forest	09/06/2008
30/01/2012	Male	5	St Martins	31/05/2006
03/02/2012	Female	1	Vale	05/12/2010
06/02/2012	Female	4	Vale	07/03/2007
08/02/2012	Male	10	St Peter Port	04/03/2001
12/02/2012	Male	1	St Peter Port	12/08/2010
21/02/2012	Female	44	Castel	23/06/1967
24/02/2012	Male	43	St Peter Port	13/07/1968
27/02/2012	Male	2	St Peter Port	08/08/2009
27/02/2012	Male	1	St Peter Port	12/08/2010
09/03/2012	Female	7	St Sampsons	03/01/2005
09/03/2012	Male	0	St Martins	22/03/2011
10/03/2012	Male	0	St Peter Port	07/07/2011

12/03/2012	Male	3	Forest	09/06/2008
12/03/2012	Female	19	St Sampsons	14/05/1992
14/03/2012	Female	42	St Martins	08/04/1969
20/03/2012	Male	0	St Peter Port	14/07/2011
22/03/2012	Female	3	St Martins	27/10/2008
13/04/2012	Female	77	Castel	27/07/1934
29/04/2012	Male	0	St Peter Port	14/07/2011
10/05/2012	Female	1	St Peter Port	24/02/2011
18/05/2012	Female	64	Vale	04/09/1947
25/05/2012	Female	43	St Peters	11/09/1968
12/06/2012	Female	3	St Peter Port	18/10/2008
13/06/2012	Female	21	St Peter Port	03/01/1991
15/06/2012	Male	1	St Peter Port	12/08/2010
18/06/2012	Male	4	Forest	09/06/2008
24/06/2012	Male	2	St Peter Port	23/12/2009
05/07/2012	Male	2	Vale	27/05/2010
06/07/2012	Male	3	St Andrews	24/06/2009
08/07/2012	Female	3	St Peter Port	19/02/2009
10/07/2012	Female	67	Castel	12/05/1945
10/07/2012	Male	4	St Saviours	04/11/2007
10/07/2012	Male	4	Castel	05/05/2008
12/07/2012	Male	4	Vale	16/06/2008
12/07/2012	Female	3	St Andrews	04/09/2008
14/07/2012	Male	2	Alderney	06/10/2009
16/07/2012	Female	36	St Peter Port	02/01/1976
06/08/2012	Female	21	St Peter Port	03/01/1991
13/08/2012	Male	2	Vale	27/05/2010
27/08/2012	Female	5	Castel	06/12/2006
03/09/2012	Male	2	Vale	27/05/2010
14/09/2012	Male	2	Vale	03/01/2010
24/09/2012	Female	52	Vale	24/07/1960
24/09/2012	Female	22	St Andrews	27/02/1990
24/09/2012	Male	2	Vale	27/05/2010
01/10/2012	Female	7	Forest	11/11/2004
02/10/2012	Male	3	Castel	17/10/2008
03/10/2012	Female	22	St Peter Port	02/08/1990
04/10/2012	Female	13	Forest	04/05/1999
10/10/2012	Female	2	St Martins	04/07/2010
12/10/2012	Male	8	Castel	01/09/2004
15/10/2012	Male	1	St Andrews	10/10/2011
23/10/2012	Female	0	Alderney	01/01/2012
24/10/2012	Male	1	St Peter Port	11/05/2011
27/10/2012	Female	1	St Peter Port	15/11/2010
12/11/2012	Female	14	St Sampsons	30/05/1998
21/11/2012	Female	0	Castel	29/03/2012
22/11/2012	Male	76	Vale	17/06/1936

28/11/2012 Male 11 Vale 03/05/2001

Asthma admissions - 2008-2012

Year and gender

Gender	2008	2009	2010	2011	2012
Female	44	30	29	34	32
Male	34	39	23	24	31
Grand Total	**78**	**69**	**52**	**58**	**63**

Year and age

Age	2008	2009	2010	2011	2012
0	3	1		2	6
1	8	8	3	8	8
2	12	6	11	9	9
3	10	8	4	4	8
4	8	7	4	4	6
5	1	5	2	5	2
6		3		1	
7	3	2	1		2
8	2	1			1
9	1	1	1	1	
10	3	1	2		1
11	2	1	1		1
12	3	1	1	1	
13		3			1
14		2			1
15	1		1		
17				1	
18	3		2		
19			2		1
20			2	3	
21				2	3
22					2

23		1		1	
26	1				
27	1				
28		1	1		1
29			1	1	
32	1	2			
33	1				
34		1			
36					1
38			1	1	
39	1		1		
41	1	1			
42	1	1			1
43	1	1			2
44			3		1
46	1	1	1	2	
47				1	
48				1	
49		1			
50	1				
52		1			1
53	1	1			
54		2			
55				1	
56				1	
58	1				
60				1	
61		1		1	
63	2				
64		2	1	1	1
65			1		
67					1
68			1		
71		1			
72				1	
73			1		
76			2	1	1
77					1
80				1	
83	1		1	1	

84	3	1			
87				1	
Grand Total	**78**	**69**	**52**	**58**	**63**

Year and parish

PARISH	2008	2009	2010	2011	2012
Alderney		1			2
Castel	15	8	6	6	8
Forest	1	1	2	4	5
Herm			1		
No address			1		
Sark			1	2	
St Andrews	6		2	2	4
St Martins	6	6	6	4	5
St Peter Port	20	21	14	23	20
St Peters	1	1			1
St Sampsons	15	8	4	5	3
St Saviours	2	4	5	1	3
Torteval	2	2	1	1	
Vale	10	17	9	10	12
Grand Total	**78**	**69**	**52**	**58**	**63**

Year and number of patient attendances

URN	2008	2009	2010	2011	2012
1549	1				
8403	1				
10516	3				
10977	1				
17227	1				
22721	1				
27017	1				
30857	1	2			
33784		1			
33828		1		1	
43591	1	2			
43889		1			
45490		1			1

45897	1				
46954	1		1		
50040	1				
51843		1			
53495		1			1
59180		1			
59533	1		2	1	
63126	1				
63689	1				
65502	3		1		1
68312	1				
69769	1				
75697		1			
93739		1			
95455	2	2			
98474	2				
99436		1			
101911		1	1		1
103435	1				
104208	1				
104698	1				
109430		1			
109998	1				
111952		1			
113486	1	1	1		
117786	1				
122061		1			
122888		1			
125317		1			
125960		1	1		
126807		1			
127516		1			
128387		1			
128540	1				
128962	1				
129987	1				
130124	1				
130545		2			
130625	1				1
130810	1				

130898		1			
131251	1				
131517	1			1	
131820	2				1
131822	1				
132354	1				
132551		2	3		
132633	2				
132660		1			
132690	1				
134712	2				
134822	2	1			
135052		1			
135583		1			
135832	1				
135944	1	1			
136405	2	1			
136453	1				
136639	4			2	
137631		1			
138204	2				
138308	1	1			
138533		1			
139607	1				
139820	1		2		
139999	1				
140656	1				
141235	3				
141526	1				
141688	1				
141843	1			1	
142020		1			
142315		1			
142378		1		1	
142399		1			
142669	1		1		
142992	1				
143042		1			
143118	1				
143692		1			

143918		1			
144031		1			
144918		2			
145504	1				
145673	1				
145859		3			
148776		1			
200613				1	
302863				1	
201114					1
200885					1
201128					2
201441					1
201596					1
143885			2	3	3
146014			2		
200117				2	1
147709				1	
200098				1	
148852				1	
147328				1	
148772				1	
200421				1	1
200719					1
200505					3
200849					1
201290					1
200981					1
200682					1
141845		1			
139520		1			
140150			2		
142234			1		
142658			2		
143594			1	1	
143441			1		
302123			1		
142415			1		
146378				1	
145376				3	1

146139				1	
145384				1	
145435				1	
148029					1
200355					4
148640					1
200133					1
137620		1			
135270		1			
135271		1			
139819			1	1	1
140128			1		
145444					1
147554					1
146390					1
304957					1
145367					1
133248		1			
131841		1			
136195			1		
135967			1		
300333				1	
138999				1	
140146				1	
139521					1
143508					1
142025					1
143966					1
130260		1			
131759			1		
139743				1	
135993				1	
148695				1	
136865					1
138823					1
303601					1
119595		1			
119371			1		
119400				1	
107461			1		

116477					1
117107					1
96754			1		
110036				1	
107856					1
88843			1		
73359				1	
90614			1		
302390			1		1
71592			1	3	2
63586			1		
61720			1		
63061				1	
86160				1	2
86761				1	
304194					1
40225			1		
50850				1	
146350					1
99454			1		
125401				1	
138326			1		
46328					1
112841					1
12278			1		
51444					1
26302		1		1	
57967			1		
15964				2	
116927		1			
33808				1	
62937				1	
44184				1	
31341		1			
991		1			
13895			1		
10676				1	
53017					1
2331			1		
5328					1

6511			1		
82807				1	
26782			1		
48132			1		
82433			1		
11127				1	
58365					1
19017					1
11145				1	
22877			1		
43515				1	
44381		1			
18344				1	
Grand Total	**78**	**69**	**52**	**58**	**63**

A3.5 Hospital admissions per parish, standardised rates for 2008-2012

Parish	Population	Number of admissions 2008	Number per 10000
Castel	9467	15	15.84451252
Forest	1634	1	6.11995104
St Andrew	2541	6	2.361275089
St Martin	6610	6	9.077155825
St Peter Port	17392	20	11.49954002
St Pierre du Bois	2308	1	4.332755633
St Sampson	9063	15	16.55081099
St Saviour	2844	2	7.032348805
Torteval	1026	2	19.49317739
Vale	10098	10	9.902951079

Parish	Population	Number of admissions 2009	Number per 10000
Castel	9467	8	8.450406676
Forest	1634	1	6.11995104
St Andrew	2541	0	0
St Martin	6610	6	9.077155825
St Peter Port	17392	21	12.07451702
St Pierre du Bois	2308	1	4.332755633
St Sampson	9063	8	8.827099195
St Saviour	2844	4	14.06469761
Torteval	1026	2	19.49317739
Vale	10098	17	16.83501684

Parish	Population	Number of admissions 2010	Number per 10000
Castel	9467	6	6.337805007
Forest	1634	2	12.23990208
St Andrew	2541	2	0.787091696
St Martin	6610	6	9.077155825
St Peter Port	17392	14	8.049678013
St Pierre du Bois	2308	0	0
St Sampson	9063	4	4.413549597
St Saviour	2844	5	17.58087201
Torteval	1026	1	9.746588694
Vale	10098	9	8.912655971

Parish	Population	Number of admissions 2011	Number per 10000
Castel	9467	6	6.337805007
Forest	1634	4	24.47980416
St Andrew	2541	3	1.180637544
St Martin	6610	4	6.051437216
St Peter Port	17392	23	13.22447102
St Pierre du Bois	2308	0	0
St Sampson	9063	5	5.516936997
St saviour	2844	1	3.516174402
Torteval	1026	1	9.746588694
Vale	10098	10	9.902951079

Parish	Population	Number of admissions 2012	Number per 10000
Castel	9467	8	8.450406676
Forest	1634	5	30.5997552
St Andrew	2541	4	1.574183392
St Martin	6610	5	7.56429652
St Peter Port	17392	20	11.49954002
St Pierre du Bois	2308	1	4.332755633
St Sampson	9063	3	3.310162198
St saviour	2844	3	10.54852321
Torteval	1026	0	0
Vale	10098	12	11.8835413

A3.6 Descriptive statistics for calculations

Summary Output for Regression Analysis between 'Mean Nitrogen Dioxide Level (ppb)' and 'Number of Hospital Admissions' for 2008

SUMMARY OUTPUT								
Regression Statistics								
Multiple R	0.067241767							
R Square	0.004521455							
Adjusted R Square	-0.095026399							
Standard Error	3.66591876							
Observations	12							
ANOVA								
	df	*SS*	*MS*	*F*	*Significance F*			
Regression	1	0.610396459	0.610396459	0.045419917	0.835515763			
Residual	10	134.3896035	13.43896035					
Total	11	135						
	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*	*Lower 95.0%*	*Upper 95.0%*
Intercept	5.350934879	5.494522189	0.973867189	0.353090696	-6.891623483	17.59349324	-6.891623483	17.59349324
Mean No2	0.106526433	0.499843694	0.213119489	0.835515763	-1.007194721	1.220247587	-1.007194721	1.220247587

Summary Output for Regression Analysis between 'Mean Nitrogen Dioxide Level (ppb)' and 'Number of Hospital Admissions' for 2009

SUMMARY OUTPUT								
Regression Statistics								
Multiple R	0.005170024							
R Square	2.67291E-05							
Adjusted R Sq	-0.099970598							
Standard Error	3.265942675							
Observations	12							
ANOVA								
	df	*SS*	*MS*	*F*	*Significance F*			
Regression	1	0.002851109	0.002851109	0.000267299	0.987277349			
Residual	10	106.6638156	10.66638156					
Total	11	106.6666667						
	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*	*Lower 95.0%*	*Upper 95.0%*
Intercept	5.740470466	4.611597189	1.244790087	0.241590779	-4.5348084	16.01574933	-4.5348084	16.01574933
mean no2	-0.006842661	0.418530088	-0.016349269	0.987277349	-0.939385811	0.925700489	-0.939385811	0.925700489

Summary Output for Regression Analysis between 'Mean Nitrogen Dioxide Level (ppb)' and 'Number of Hospital Admissions' for 2010

SUMMARY OUTPUT								
Regression Statistics								
Multiple R	0.35251071							
R Square	0.1242638							
Adjusted R Square	0.03669018							
Standard Error	1.731876579							
Observations	12							
ANOVA								
	df	*SS*	*MS*	*F*	*Significance F*			
Regression	1	4.256035162	4.256035162	1.418963843	0.261079593			
Residual	10	29.99396484	2.999396484					
Total	11	34.25						
	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*	*Lower 95.0%*	*Upper 95.0%*
Intercept	1.908171792	2.028510275	0.940676424	0.369038081	-2.611630763	6.427974346	-2.611630763	6.427974346
mean no2	0.166985195	0.140182017	1.191202688	0.261079593	-0.145359803	0.479330193	-0.145359803	0.479330193

Summary Output for Regression Analysis between 'Mean Nitrogen Dioxide Level (ppb)' and 'Number of Hospital Admissions' for 2011

SUMMARY OUTPUT								
Regression Statistics								
Multiple R	0.552865172							
R Square	0.305659899							
Adjusted R Square	0.236225889							
Standard Error	1.780679588							
Observations	12							
ANOVA								
	df	*SS*	*MS*	*F*	*Significance F*			
Regression	1	13.95846872	13.95846872	4.402163989	0.06227229			
Residual	10	31.70819795	3.170819795					
Total	11	45.66666667						
	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*	*Lower 95.0%*	*Upper 95.0%*
Intercept	0.064027344	2.330515553	0.027473468	0.978622645	-5.128684903	5.256739592	-5.128684903	5.256739592
mean no2	0.377442933	0.179894626	2.098133454	0.06227229	-0.023387272	0.778273138	-0.023387272	0.778273138

Summary Output for Regression Analysis between 'Mean Nitrogen Dioxide Level (ppb)' and 'Number of Hospital Admissions' for 2012

SUMMARY OUTPUT								
Regression Statistics								
Multiple R	0.350143565							
R Square	0.122600516							
Adjusted R Square	0.034860568							
Standard Error	3.110197632							
Observations	12							
ANOVA								
	df	*SS*	*MS*	*F*	*Significance F*			
Regression	1	13.5167069	13.5167069	1.397316939	0.264518893			
Residual	10	96.7332931	9.67332931					
Total	11	110.25						
	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*	*Lower 95.0%*	*Upper 95.0%*
Intercept	-4.089298343	7.951573477	-0.514275364	0.618232447	-21.80650814	13.62791146	-21.80650814	13.62791146
mean no2	0.677783974	0.573381708	1.182081613	0.264518893	-0.599790086	1.955358035	-0.599790086	1.955358035

Appendix 4

A4.1 Participant Information letter

A4.2 Participant consent form
A4.3 Participant Survey form

A4.1 Participant Information Letter

HEALTH AND SOCIAL SERVICES
A STATES OF GUERNSEY GOVERNMENT DEPARTMENT

UNIVERSITY of ABERTAY DUNDEE

Participant Information Letter **November 2013**

PhD research project :

Indoor environment and the impacts on the health of pre-existing asthmatics at work – the development of a risk management framework.

Dear……..

I am undertaking some research into the effects of indoor air pollution on people suffering with asthma and would like to ask you some questions that will assist with this research.

The research is looking at the effects of nitrogen dioxide, an irritant gas, on the health of people with asthma. Nitrogen dioxide is a gas associated with vehicle emissions, boilers, open fires, gas cookers etc. and is known to exacerbate symptoms in those already suffering with asthma.

The data gathered may also be used to assist with a new States report on air pollution control measures due to be debated by the States next year.

I do not need your name, address or date of birth so any information you provide will be anonymous.

I attach the questionnaire to this letter and hope that you will complete it and leave it with Rebecca Sherrington, Respiratory Nurse Consultant, MSG.

Thank you for assisting with this project.

Yours sincerely

Valerie Cameron

Director of Environmental Health and Pollution Regulation.

A4.2 Participant consent form

HEALTH AND SOCIAL SERVICES
A STATES OF GUERNSEY GOVERNMENT DEPARTMENT

UNIVERSITY of ABERTAY DUNDEE

November 2013

PhD research project :
Indoor environment and the impacts on the health of pre-existing asthmatics at work – the development of a risk management framework

CONSENT FORM

Researchers **Valerie Cameron** **Rebecca** **Sherrington**		**Please initial box**
1. I confirm that I have read and have understood the Participant Information Letter dated November 2013 for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.		☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason, without my rights being affected.		☐
3. I agree to take part in the above study.		☐

Participant Name	Date	Signature
Name of Person taking consent	Date	Signature
Researcher	Date	Signature

The contact details of lead Researcher are:

Valerie Cameron,
Director of Environmental Health and Pollution Regulation,
Longue Rue, St Martin. Tel 711161 Email: vcameron@hssd.gov.gg

A4.3 Participant Survey form

Asthma Case Study Questionnaire November 2013

Post code…….Gender M/F……Age…….
Workplace
Postcode……
Do you smoke?.Y\N…..Does anyone in your household smoke?.....Y\N……
Do you have pets living in your house?.....Y\N…If yes, what type?.....
Do you suffer from allergies? Y\N….If yes, what kind?.....…….....

1 Have you experienced wheeze or tightness in the chest this month?
Yes
No
If no, questionnaire completed. Thank you.

If yes, please mark the date(s) on the following grid.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	

2 Have any of the episodes required you to increase your usual medication?
Yes
No
If yes, please mark the date(s) on the following grid.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	

3 Have any of the episodes required hospital treatment/admission?
Yes
No
If yes, please mark the date(s) on the following grid

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	

4 What do you think caused your asthma symptoms to get worse?
.....
Thank you for completing this questionnaire.

Appendix 5

A5.1 Descriptive statistics for calculations

A5.1 Descriptive statistics for calculations

No2 Levels at 300mm (ppb)	
Mean	4.884848485
Standard Error	0.194226737
Median	4
Mode	3
Standard Deviation	2.494887603
Sample Variance	6.224464154
Kurtosis	0.254428148
Skewness	1.04847294
Range	12
Minimum	1
Maximum	13
Sum	806
Count	165

NO2 Levels at 600mm (ppb)	
Mean	4.860606061
Standard Error	0.19526868
Median	4
Mode	3
Standard Deviation	2.508271608
Sample Variance	6.29142646
Kurtosis	0.517268573
Skewness	1.12918269
Range	12
Minimum	1
Maximum	13
Sum	802
Count	165

NO2 Levels at 900mm (ppb)	
Mean	4.575757576
Standard Error	0.179954728
Median	4
Mode	3
Standard Deviation	2.31156033
Sample Variance	5.34331116
Kurtosis	0.605815335
Skewness	1.170318125
Range	11
Minimum	1
Maximum	12
Sum	755
Count	165

NO2 Levels at 1200mm (ppb)	
Mean	4.927272727
Standard Error	0.197277036
Median	4
Mode	3
Standard Deviation	2.534069407
Sample Variance	6.421507761
Kurtosis	-0.25398144
Skewness	0.956563783
Range	11
Minimum	1
Maximum	12
Sum	813
Count	165

NO2 Levels at 1500mm (ppb)	
Mean	4.787878788
Standard Error	0.188898835
Median	4
Mode	3
Standard Deviation	2.426449476
Sample Variance	5.887657058
Kurtosis	-0.087345323
Skewness	1.010589829
Range	11
Minimum	1
Maximum	12
Sum	790
Count	165

Summary Output for Regression Analysis between 'Distance from wall (mm)' and 'Mean NO2 Level (ppb)'

SUMMARY OUTPUT								
Regression Statistics								
Multiple R	0.90530725							
R Square	0.819581218							
Adjusted R Square	0.799534686							
Standard Error	1.598534683							
Observations	11							
ANOVA								
	df	*SS*	*MS*	*F*	*Significance F*			
Regression	1	104.4712727	104.4713	40.88394	0.000126151			
Residual	9	22.99781818	2.555313					
Total	10	127.4690909						
	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*	*Lower 95.0%*	*Upper 95.0%*
Intercept	12.15636364	1.033724089	11.75978	9.15E-07	9.817917285	14.49480999	9.817917285	14.49480999
Distance from wall (mm)	-0.003248485	0.000508048	-6.39406	0.000126	-0.004397769	-0.002099201	-0.004397769	-0.002099201

Mean NO^2 level at distance from wall along transect from open fireplace

Distance from wall (mm)	Mean NO2 Level (ppb)
300	12
600	11
900	10.4
1200	8.8
1500	6.6
1800	3.2
2100	3.8
2400	3.2
2700	3.4
3000	3.2
3300	3.8

Appendix 6

A6.1 Questionnaire to EHOs and Authorised Officers in Scotland

A6.2 Access Database printout - Survey responses

A6.3 Letter to Society of Chief Officers of Environmental Health In Scotland and reply

A6.1 Questionnaire to EHOs and Authorised Officers in Scotland

Questionnaire to Authorised Officers

Any information supplied in response to this questionnaire will be completely confidential. All data will be stored in an Access database and will be entirely ring-fenced.

If you wish, your employing authority can be acknowledged as contributing to this research, please state below.

Employing Authority.....(if acknowledgement requested)

1 Your employment – What is your Health and Safety enforcement role.

Are you employed as:-

Head of Service ☐

Health and Safety at Work Manager ☐

Health and Safety Enforcement Officer ☐

Environmental Health Officer – HSW full time ☐

Environmental Health Officer – including HSW ☐

Other.....

2 Has your authority developed an operational partnership agreement?

Yes ☐

No ☐

3 Did your authority previously take part in the Fit3 programme?

Yes ☐

No ☐

4 Does your authority undertake 'themed' or 'topic-based' inspections?

Yes ☐

No ☐

5 Do you think themed/topic-based inspections are effective in managing HSW risk?

Yes ☐

No ☐

6 If 'yes' to question 3, did your authority give any priority to the three DRP priorities?

Asthma	Yes	☐	No	☐
Dermatitis	Yes	☐	No	☐
Asbestos	Yes	☐	No	☐

7 Have you continued to give priority to the DRP topics under OPA?

Yes ☐

No ☐

Asthma

8 Do you think you have enough technical knowledge to assess risk to pre-existing asthmatics in workplaces?

Yes ☐

No ☐

9 Which of the following hazards do you think affect pre-existing asthmatics at work?

(You may tick more than one)

Dust ☐

Fume ☐

High Humidity ☐

Low Humidity ☐

High temperature ☐

Low temperature ☐

Steam ☐

10 Which categories of workplace do you think would impact on pre-existing asthmatics?

Restaurants ☐

Stonemason ☐

Dry cleaners ☐

Hot Food Take-away ☐

Banks ☐

Offices ☐

Paint spraying ☐

Warehouses ☐

Tyre and exhaust ☐

Bakery ☐

Fuel service station ☐

Hairdressers ☐

11 After an inspection, do you consider asthma, when calculating the HSW priority rating score for health or safety? (Please be honest)

Yes ☐

No ☐

If yes, in what way do you consider asthma in your calculation of the HSW priority rating score?

Thank you for completing this questionnaire. Your reply is CONFIDENTIAL.

A6.2 Access Database printout - Survey responses

Responses 22/05/2011

ID	Q01EmployedAs	Q01EmployedAsOther	Q02OperationalPart
549	EHO		☐
548	EHO		☑
547	EHO		☑
546	EHO-HSW		☐
545	HSWM		☑
544	EHO-HSW		☑
543	HSEO		☑
542	EHO		☑
541	HoS		☑
540	HSEO		☐
539	EHO		☐
538	HSWM		☑
1	EHO		☑

Responses 22/05/2011

Q03Fit3	Q04ThemedInspecti	Q05AreEffective	Q06 Asthma
☑	☑	☐	☑
☑	☑	☑	☐
☑	☑	☑	☐
☑	☑	☑	☑
☑	☑	☑	☐
☑	☑	☑	☐
☑	☑	☑	☐
☑	☑	☑	☑
☑	☑	☑	☑
☑	☑	☑	☑
☑	☑	☑	☑
☑	☑	☑	☐
☑	☑	☑	☑

Responses 22/05/2011

Q06Dermatitis	Q06 Asbestos	Q07ContinuedP	Q08EnoughKnowle	Q09 Dust
☑	☑	☑	☐	☑
☑	☐	☑	☐	☑
☑	☐	☑	☑	☑
☑	☑	☐	☑	☑
☑	☑	☑	☐	☑
☑	☑	☐	☐	☑
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☑	☑	☐	☐	☑
☑	☑	☐	☑	☑
☑	☐	☑	☐	☑

Responses 22/05/2011

Q09 Fume	Q09 HumidityHigh	Q09 HumidityLow	Q09 TemperatureHigh
☑	☑	☑	☑
☑	☑	☑	☑
☑	☑	☑	☑
☑	☐	☐	☐
☑	☐	☐	☐
☑	☐	☑	☐
☑	☑	☑	☑
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☑	☑	☑	☑
☑	☐	☐	☐
☑	☑	☑	☐
☑	☐	☐	☐
☑	☑	☐	☐

Responses 22/05/2011

Q09TemperatureLow	Q09 Steam	Q10 Restaurants	Q10 Stonemason
☑	☑	☑	☑
☑	☑	☑	☑
☑	☐	☑	☑
☐	☐	☐	☑
☐	☐	☑	☑
☑	☐	☑	☑
☑	☐	☑	☑
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☐	☐	☑	☑
☐	☑	☐	☑
☑	☐	☐	☑
☑	☑	☐	☑

Responses 22/05/2011

Q10 PaintSpraying	Q10 Warehouses	Q10TyreAndExhaust	Q10 Bakery
☑	☑	☑	☑
☑	☐	☑	☑
☑	☐	☑	☑
☑	☐	☐	☐
☑	☑	☑	☑
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☑	☐	☑	☑
☑	☐	☑	☑
☑	☐	☑	☑

A6.3 Letter to Society of Chief Officers of Environmental Health In Scotland and reply

Copy

2 Annfield Farm Cottage
Dunfermline
Fife
KY11 7EU

17th October 2006

Mr D Evans
Chairman, Society of Chief Officers of Environmental Health in Scotland
Environmental Health and Consumer Services
East Lothian Council
John Muir House
Haddington
EH41 4RP

Dear David,

PhD Research into Asthma at Work

Further to our telephone conversation, I confirm that I am undertaking research into the effect of the indoor environment on pre-existing asthmatics at work leading to the development of a risk management framework.

As part of my research, I would like to send a questionnaire to Health and Safety Enforcement Officers based in Scottish local authorities, probably during 2007.

I would be very grateful if you could consider this proposal and provide a letter of support from the Society for this research.

Kindest regards.

Yours sincerely

Valerie Cameron

SOCIETY OF CHIEF OFFICERS OF ENVIRONMENTAL HEALTH IN SCOTLAND

6 November 2006

Valerie Cameron
2 Annfield Farm Cottage
DUNFERMLINE
Fife
KY11 7EU

Dear Val

PhD Research into Asthma at Work

I refer to your letter of 17 October 2006 and would confirm that I see no difficulty in the Society supporting your research by way of a questionnaire to Scottish Local Authority Health & Safety Enforcement Officers in 2007.

Best wishes for your studies.

Yours sincerely

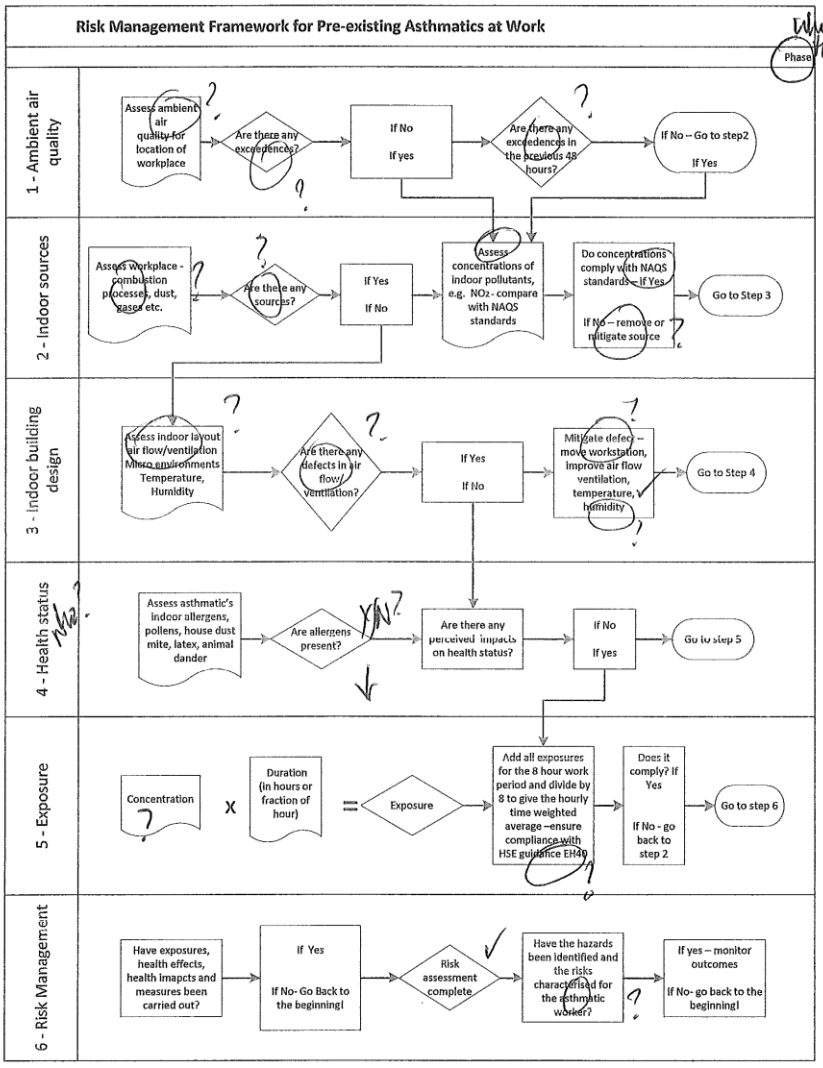
David Evans

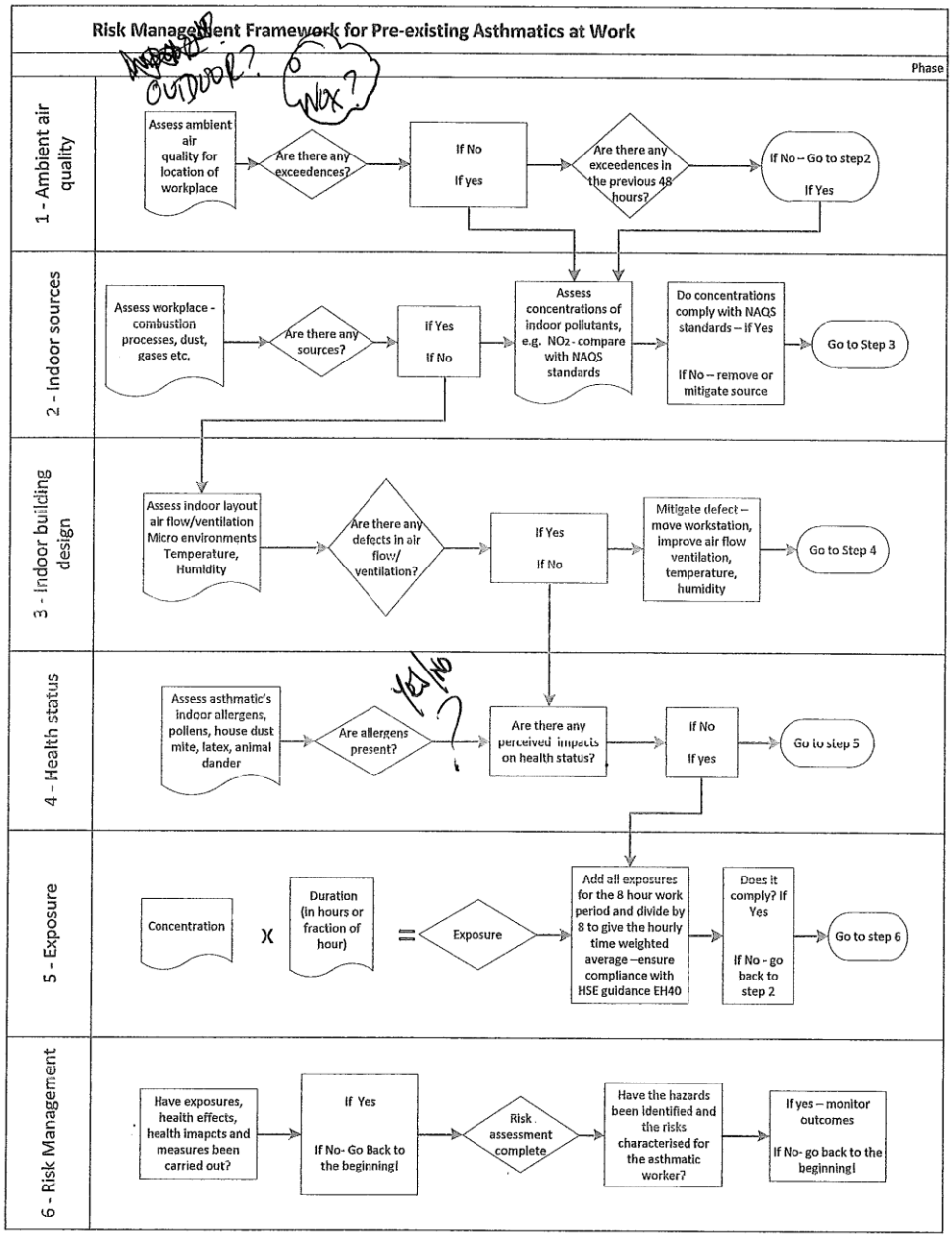
David K Evans
Chairman, Society of Chief Officers of Environmental Health In Scotland
C/o East Lothian Council
Environmental & Consumer Services
John Muir House
Haddington
EH41 3HA

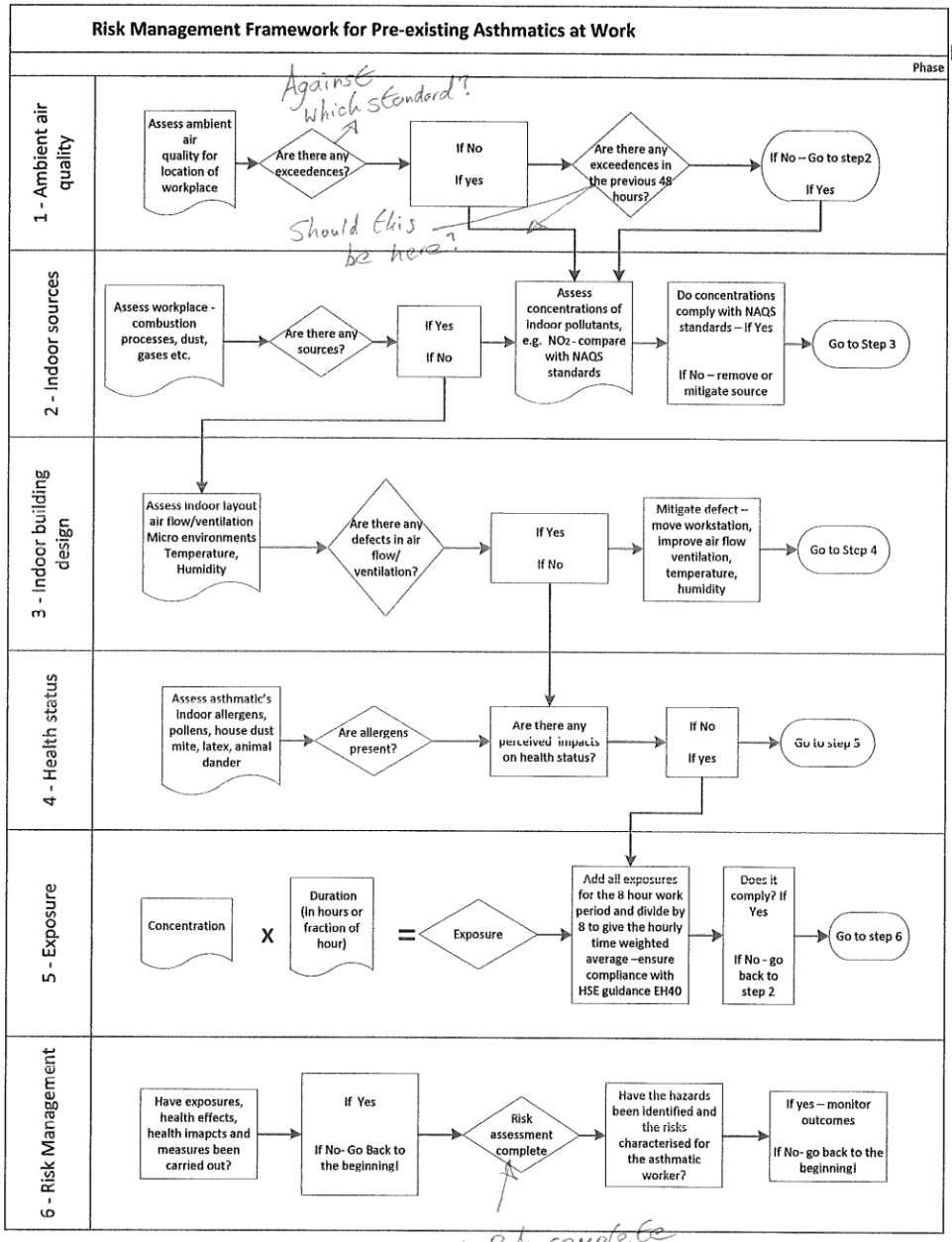
Appendix 7

A7.1 Responses to draft risk management framework calibration exercise

A7.1 Responses to draft risk management framework calibration exercise




Risk Management Framework for Pre-existing Asthmatics at Work
Phase
1 - Ambient air quality
Assess ambient air quality for location of workplace
Are there any exceedences?
If No
If yes
Are there any exceedences in the previous 48 hours?
If No – Go to step2
If Yes
2 - Indoor sources
Assess workplace - combustion processes, dust, gases etc.
Are there any sources?
If Yes
If No
Assess concentrations of indoor pollutants, e.g. NO2 - compare with NAQS standards
Do concentrations comply with NAQS standards – If Yes
If No – remove or mitigate source
Go to Step 3
3 - Indoor building design
Assess indoor layout air flow/ventilation Micro environments Temperature, Humidity
Are there any defects in air flow/ ventilation?
If Yes
If No
Mitigate defect – move workstation, improve air flow ventilation, temperature, humidity
Go to Step 4
4 - Health status
Assess asthmatic's indoor allergens, pollens, house dust mite, latex, animal dander
Are allergens present?
Are there any perceived impacts on health status?
If No
If yes
Go to step 5
5 - Exposure
Concentration
X
Duration (in hours or fraction of hour)
=
Exposure
Add all exposures for the 8 hour work period and divide by 8 to give the hourly time weighted average –ensure compliance with HSE guidance EH40
Does it comply? If Yes
If No - go back to step 2
Go to step 6
6 - Risk Management
Have exposures, health effects, health imapcts and measures been carried out?
If Yes
If No- Go Back to the beginning!
Risk assessment complete
Have the hazards been identified and the risks characterised for the asthmatic worker?
If yes – monitor outcomes
If No- go back to the beginning!



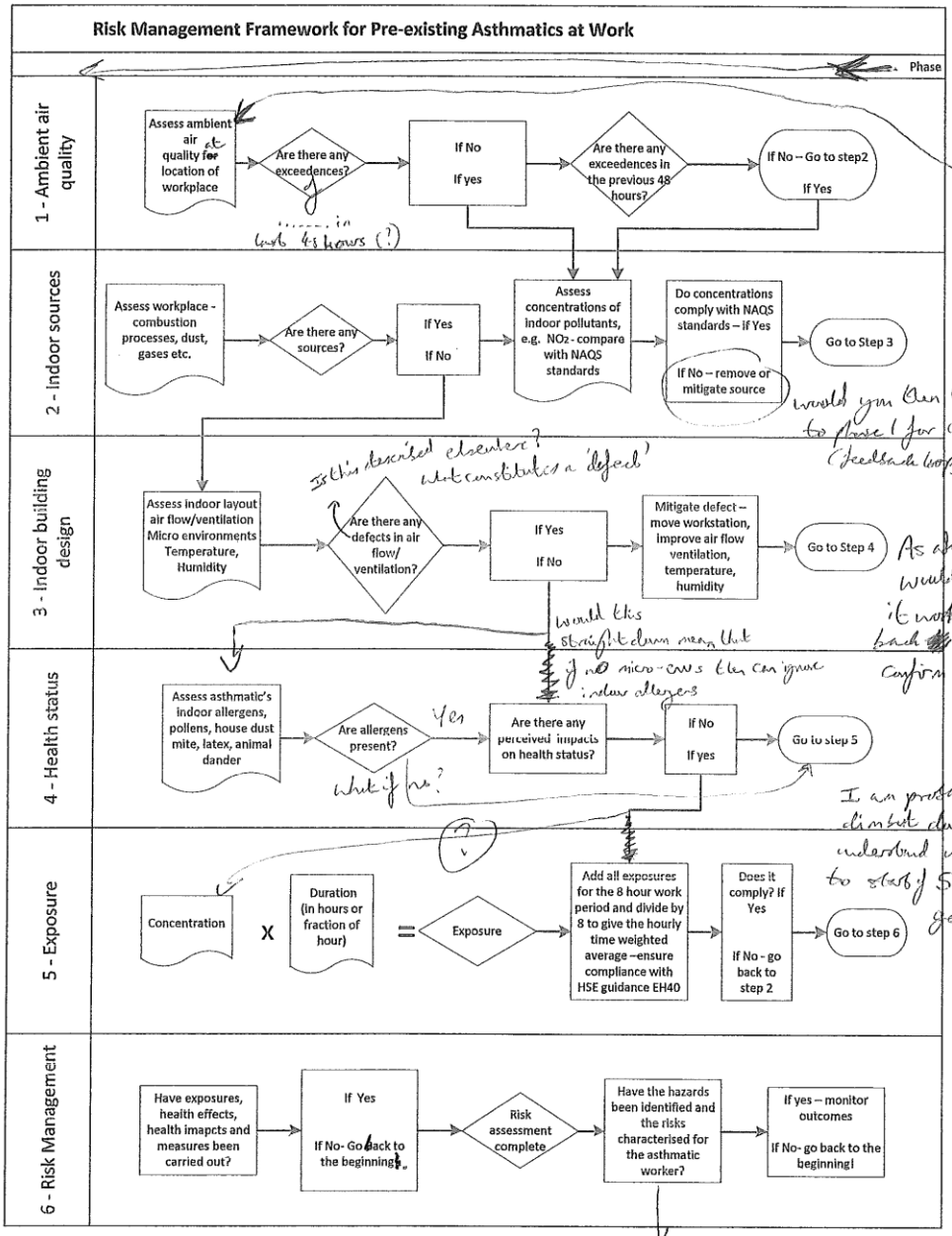
Against which standard?

What does this refer to?

Should this be here?

If RA complete why are there more steps following it?

Enforcement Officer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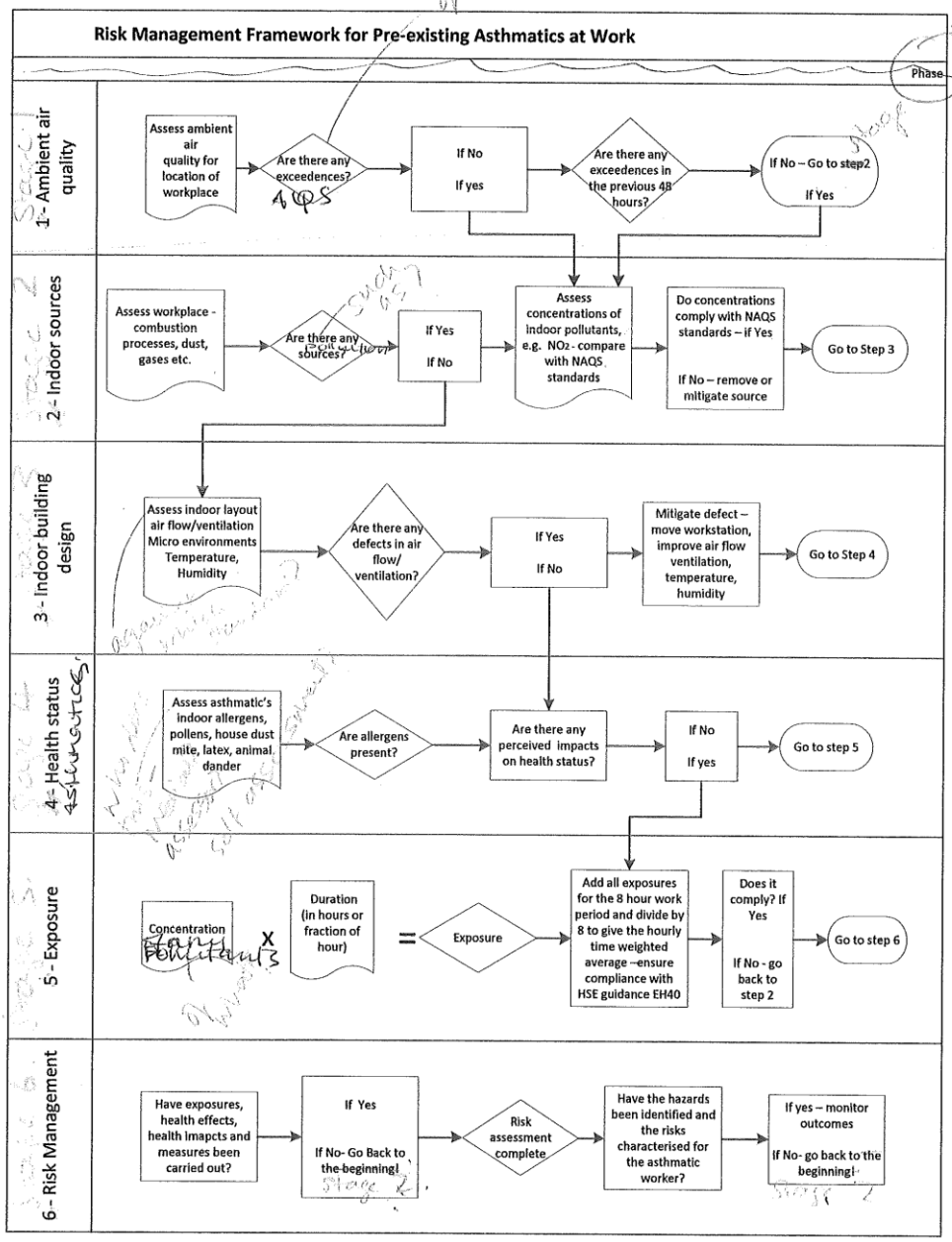
CHO1



2 EHOs on joint Inspection